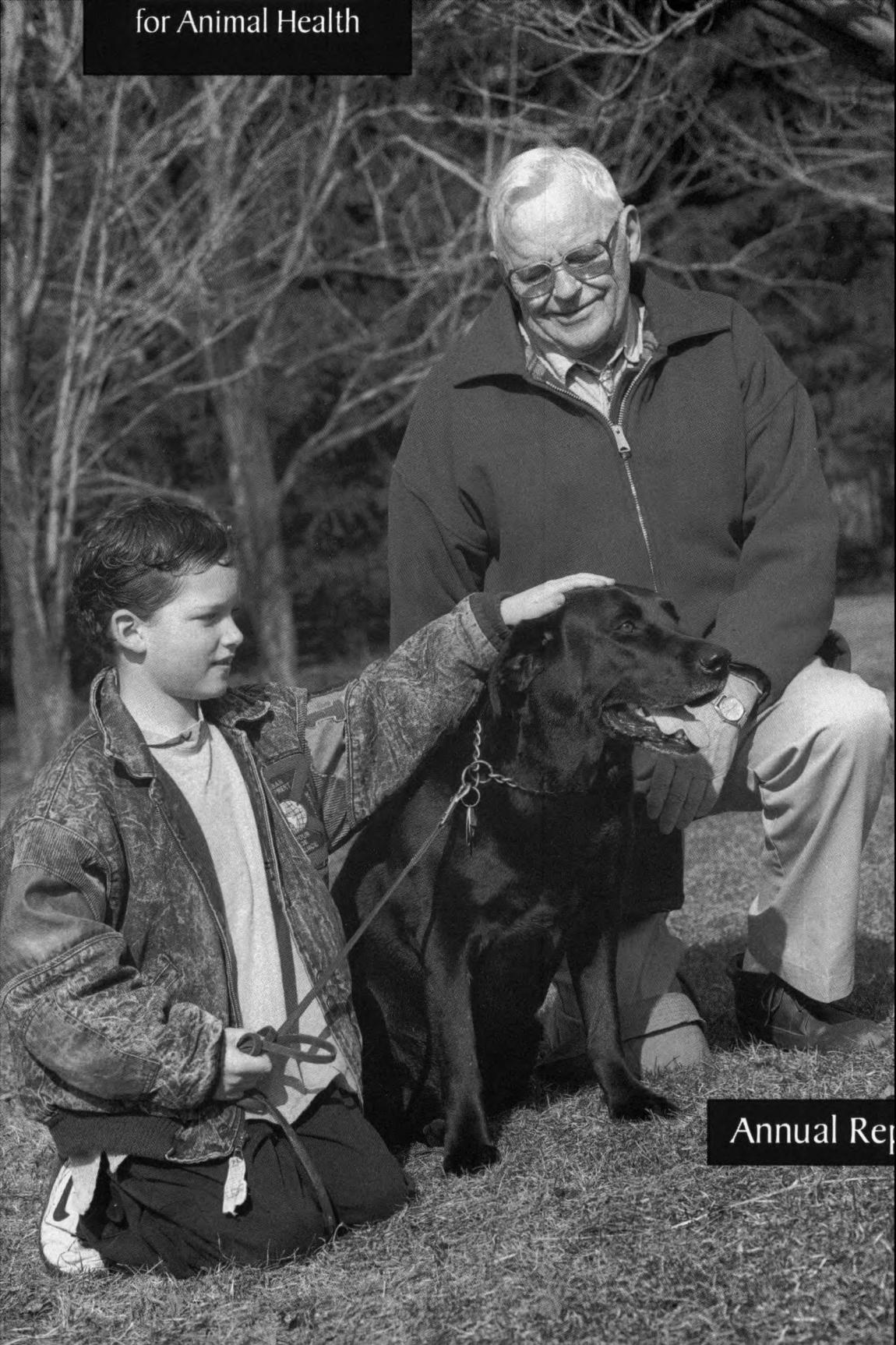


for Animal Health



Annual Rep


Annual Report, 1993, Volume 43

James A. Baker Institute
for Animal Health

College of Veterinary Medicine

Cornell University

Photography by Dede Hatch, Ithaca,
New York, unless otherwise noted.

 Printed using soy-based inks on
recycled paper with a minimum of 10
percent postconsumer wastepaper.

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Jack Hyde, D.V.M. with his friends Eiron Driscoll and Archie

A Message from the Director



ADVANCES IN BASIC ANIMAL HEALTH STUDIES are often difficult to measure in the short span of a year, but at the Baker Institute we are preparing to reap significant rewards in several areas. We are beginning to test two canine vaccines, one for Lyme disease and the other for parvovirus, that belong to a new generation of ultra-stable recombinant vaccines.

We have also developed a blood-based DNA test to detect the genetic mutation that causes progressive retinal atrophy in Irish Setters. The test can distinguish among genetically normal dogs, carriers, and those that are affected by this serious inherited eye disease. As described in the laboratory reports that follow, we have made excellent progress in many other areas as well.

Our breakthroughs in the next decade will allow us to develop tools to diagnose and treat both infectious and inherited diseases that have not yielded to traditional research methods.

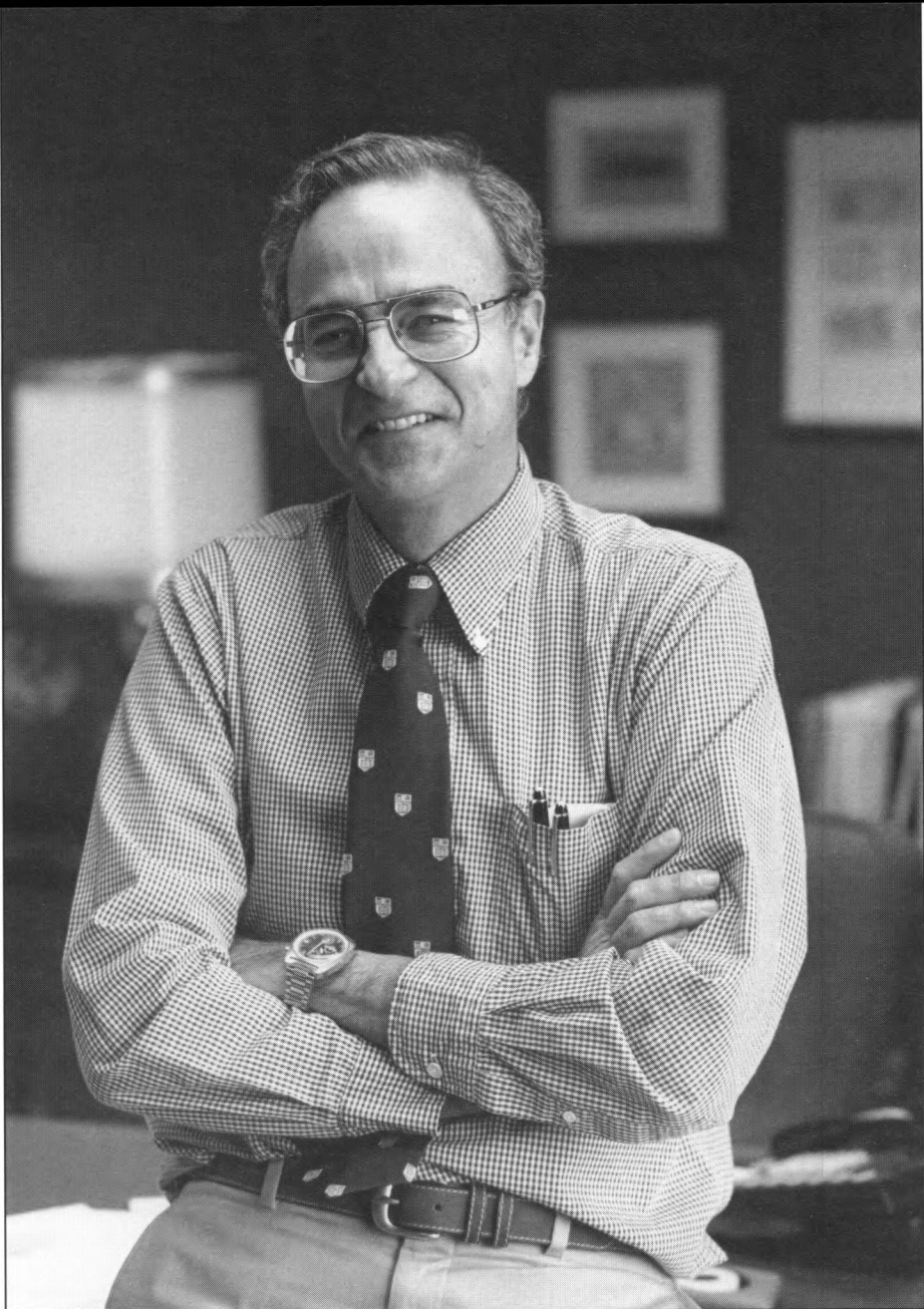
The Institute has maintained its leadership in animal health research by making full use of newly developed technologies, especially the sophisticated methods of molecular biology that are essential for today's cutting-edge work. We expect increasingly rapid growth in the complexity and promise of emerging technologies during the next few years. Many of the breakthroughs we an-

ticipate in the next decade will center on the identification of gene mutations that cause disease in dogs and other companion animals, and the characterization of

the molecular mechanisms of basic disease processes. Such information will allow us to develop tools to diagnose and treat both infectious and inherited diseases that have not yielded to traditional research methods.

While the productivity of Institute research has never been higher, public funding for companion animal research is diminishing. The National Institutes of Health (NIH), the principal source of support for biomedical research, has shifted a majority of its funding to a more limited number of areas. As a result, investigations that promise direct, major benefits for canine and equine health (and for human health as well) lack the level of funding to proceed at the pace we are prepared to set. In practical terms, this means that results that should take one or two years to achieve may take longer. For that reason, we must look increasingly to the financial support of foundations, kennel clubs, and individuals who understand the essential nature of the work we do for the sake of companion animals.

Despite these challenges, we look forward with great eagerness to the extraordinary period of discovery ahead. The Baker Institute has the superior facilities and the broad range of expertise needed



*Gus Aguirre
and Anita Hesser*

to excel in this changing research and economic climate. We have the additional benefits of an involved and knowledgeable advisory council and a cadre of loyal contributors. It is to you, our friends and benefactors, that this report is dedicated. With your help, we can continue to make a measurable difference in the quality of life we enjoy with our pets.

—Gustavo D. Aguirre



The Institute in Perspective

DURING THE 43 YEARS since its founding, the Baker Institute has been known worldwide as a leader in animal health research. From its modest beginnings as the Veterinary Virus Research Institute, it has expanded to encompass nearly a dozen areas of scientific expertise. Horses, other animals, and humans derive benefit from some of the work done here, but the Institute continues to focus primarily on health issues of concern to dog owners and breeders.

The productivity and clinical value of the research summarized in this report clearly show that the Institute remains dedicated to innovation and excellence in service to companion animal health.

Since the earliest days of the Institute, optimism and adaptability have been guiding principles.

The effectiveness of Institute scientists in reducing puppy mortality from infectious diseases has allowed concern over inherited diseases of dogs to ascend to new prominence in recent years.

While the threat of new outbreaks of contagion continues to be taken very seriously by investigators in the Cornell Research Laboratory for Diseases of Dogs, strategic planning for the Institute has also recognized the importance and the possibility of controlling hip dysplasia and other inherited canine diseases through molecular genetic research.

The establishment in early 1993 of the Center for Canine Genetics and Reproduction reflects that conviction. The

Institute has recruited seven senior investigators since 1992, gaining expertise in new disciplines and solidifying our commitment to pursuing the promise of molecular biology. Gustavo Aguirre, the director of the Center, and his colleagues in the Inherited Eye Disease Studies Unit have employed the newest technology available to develop a means to test the DNA of Irish Setters for the genetic defect that causes rod-cone dysplasia 1, a form of progressive retinal atrophy. The methods that they and other Institute faculty members are using in their research will one day be applied with equal success to the study of other inherited diseases.

Since the earliest days of the Institute, optimism and adaptability have been guiding principles. The Institute has always been fortunate to have the support of foundations, dog clubs, veterinarians, and friends who share the capacity to envision and embrace new possibilities. Their generosity assures us that the Institute can continue to lead the effort to better the lives of animals and of the people who care for them.

—Robert Shope
Chairman

ADVISORY COUNCIL

Robert E. Shope, M.D., *Chairman*

Director, Arbovirus Research Unit, Yale University

William C. Beck, M.D., F.A.C.S.

President Emeritus, Donald Guthrie Foundation for Medical Research, Sayre, Pennsylvania

Sarah R. Bogdanovitch

Lake Clear, New York

Albert C. Bostwick, Jr.

Coral Gables, Florida

Philip B. Carter, Ph.D.

Professor of Microbiology and Immunology, College of Veterinary Medicine, North Carolina State University at Raleigh

Gerald J. Chader, Ph.D., M.D. (*hc*)

Chief, Laboratory of Retinal Cell and Molecular Biology, National Eye Institute, NIH, Bethesda, Maryland

Strachan Donnelley, Ph.D.

Director of Education, The Hastings Center, Briarcliff Manor, New York

Joseph W. Jones

Chairman, The Robert W. Woodruff Foundation, Atlanta, Georgia

Patricia Kaneb

Boston, Massachusetts

Robert R. Marshak, D.V.M., D.V.M. (*hc*)

Dean Emeritus, School of Veterinary Medicine, University of Pennsylvania

Frederick A. Murphy, D.V.M., Ph.D.

Dean, School of Veterinary Medicine, University of California, Davis

Niel W. Pieper, D.V.M.*

General Practitioner, Portland, Connecticut

Gene M. Pranzo

President, The Dorothy Russell Havemeyer Foundation, New York City, New York

Henry J. Travis, D.V.M.

General Practitioner, Huntington, New York

Robert Winthrop II

Managing Director, Groton Land Company, Athens, Georgia

*Deceased

CHARLES HARRINGTON



FORMER ADVISORY COUNCIL MEMBERS

Dorothy R. Donnelley

1980–1992

Chairwoman, 1982–1988

John M. Olin

1977–1982

Gary Lee

1977–1986

Richard M. Johnson

1977–1989

G. Watts Humphrey, Jr.

1982–1989

Irwin H. Lepow, M.D.

1978

John A. Lafore, Jr.*

1978–1984

Frances G. Scaife

1978–1988

William Rockefeller

1979–1990

Chairman, 1989–1990

Robert Winthrop

1982–1990

NIEL W. PIEPER

Niel W. Pieper, D.V.M. '32, a sixteen-year member of the Institute's Advisory Council, died on September 17 in Middletown, Connecticut. Dr. Pieper practiced large and small animal medicine for fifty years in Middletown and was active in the Connecticut and American Veterinary

Medical Associations throughout his career. He received many honors in recognition of his contributions to his community and to his profession, among them the AVMA Award and Middlesex County's Outstanding Citizen Award; in 1992 the Baker Institute presented him with the Founders' Award.

A client, Priscilla Maxwell Endicott, once termed Dr. Pieper "the American equivalent of James Herriot." In his honor, Mrs. Endicott made a major bequest to Cornell University to fund Baker Institute research and scholarships for veterinary students. Dr. Pieper, a devoted advocate and supporter of the Institute's animal health programs, also made provision for the Institute in his will. We are grateful to him and to the following clients and colleagues who contributed to the Institute in his memory:

Niel Pieper in 1990, accepting a memento of the Institute's 40th anniversary celebration.

Mr. and Mrs. Raymond R. Allen

Mary C. Armstrong

Aspetuck Animal Hospital: Ferris G. Gorra, D.V.M.

Marjorie M. Cooley

Dr. and Mrs. Willard H. Daniels

Fairfield Equine Associates: Mark R. Baus, Richard D.

Mitchell, Carolyn M. Weinberg, and Ronald B. Gaeta, D.V.M.s

Fryeburg Veterinary Hospital

Margaret S. Harper

Hartford Veterinary Hospital: William A. Haines, D.V.M.

Kensington Bird and Animal Hospital: Robert F. Giddings, D.V.M.

Jane R. Lamarine

Evelyn and William E. Larson

Mr. and Mrs. Thomas McMellon

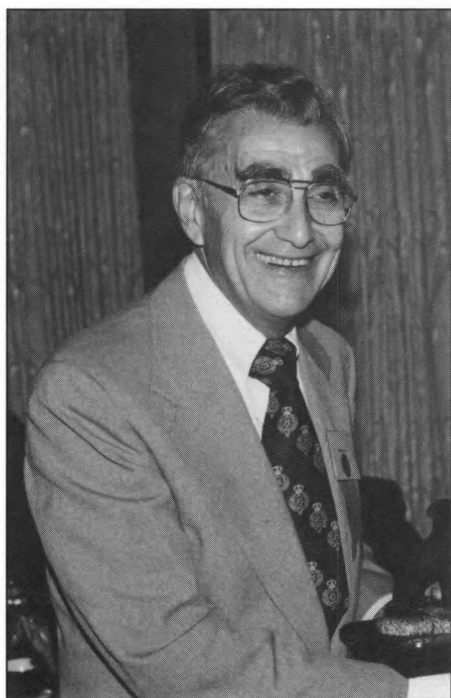
Patricia Meyer, D.V.M.

Jacqueline W. Nason

Marcia Ellen Pond

Marjorie Shenstone

Marianne and Leo J. van Dijk, D.V.M.



Above: Albert C. Bostwick, Jr., recipient of this year's Arthur F. North, Jr. Canine Service Award. Left: Harold Kopp, honored with the 1993 Founders' Award.

ANNUAL MEETING

The **Arthur F. North, Jr. Canine Service Award** was presented this year to **Albert C. Bostwick, Jr.** Mr. Bostwick, who has served on the Baker Institute's Advisory Council since 1987, was an early champion of the Institute's biotechnology ini-

tiatives. As a tribute to his late father, he and his mother, Eleanor Purviance Bostwick, sponsored the Albert C. Bostwick Laboratory of Molecular Biology. Their commitment also helped the Institute obtain other funding needed to establish leadership in the application of emerging technologies to veterinary research.

Dr. North, a 1935 graduate of Cornell's College of Veterinary Medicine, was widely known as a skilled and innovative practitioner and a strong advocate of veterinary research. The North Award recognizes those whose contributions to the Baker Institute's efforts to improve canine health and well-being reflect his spirit of concern for all dogs.

Harold Kopp, D.V.M. '42, was honored with the Institute's **Founders' Award** for 1993. Dr. Kopp is himself an Institute founder as well as a Research Partner and longtime benefactor. Before retiring from practice in 1989, Dr. Kopp

owned the Greenwich Animal Hospital in Greenwich, Connecticut, where he distinguished himself as an outstanding and tirelessly dedicated clinician.

The Founders' Award was established in 1990 in observance of the Institute's 40th anniversary. This recognition is given annually to a veterinarian whose contributions to the Institute and to his or her profession exemplify our founders' commitment to the advancement of canine medicine. The Founders'

Award has previously honored Charles Fletcher, D.V.M. '33, Du Bois Jenkins, D.V.M. '43, and Niel Pieper, D.V.M. '32.

A new award was announced at dinner following this year's Annual Meeting of the Advisory Council. The **John A. Lafore, Jr. Kennel Club Award** was established to honor Jack Lafore, a former member of the Institute's Advisory Council who died January 24th. Mr. Lafore was a noted breeder of Collies and Keeshonden. He served for many years on the American Kennel Club's Board of Directors and for eight of those years as AKC president.

The **Devon Dog Show Association** was the first club chosen to receive the Lafore Award, in recognition of their unsurpassed support of the Research Laboratory for Diseases of Dogs. The Devon Dog Show Association has contributed to the Institute since 1952. Mr. Lafore served for many years as Devon's delegate to the AKC.

NORTH AWARD RECIPIENTS

- 1982 Adelaide Riggs
- 1983 The American Kennel Club
- 1984 Priscilla Maxwell Endicott
- 1985 The Marilyn M. Simpson Charitable Trusts
- 1986 Frances Rowles Van Brunt
- 1987 The Geraldine R. Dodge Foundation
- 1988 Atherton Bristol
- 1989 Jacqueline Lindsay
- 1990 Dorothy Donnelley
- 1991 Robert Winthrop
- 1992 Eleanor Gillis

HONORABLE MENTIONS

Gustavo Aguirre was appointed an adjunct professor of ophthalmology at the University of Pennsylvania School of Medicine and Hospital. He received the Award of Merit of the Veterinary Medical Alumni Society of the University of Pennsylvania and was named to the editorial board of the journal *Investigative Ophthalmology & Visual Sciences* for a term of five years.

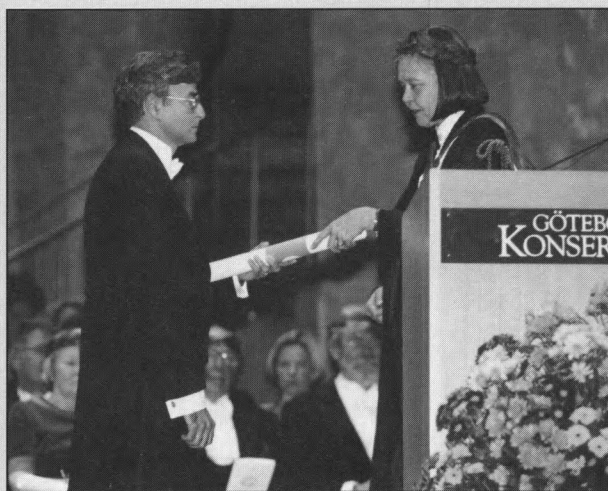
Dr. Aguirre participated in a symposium on retinal degenerations in dogs in Uppsala, Sweden and gave a lecture, "Retinal Pigment Epithelium: A Cell for all Reasons," at the University of Göteborg, Sweden. In Göteborg, he presided over the successful doctoral thesis defense of his student, Kristina Mieziwska. Dr. Aguirre spoke on progressive retinal atrophy in the dog at the Annual Meeting of the World Small Animal Veterinary Association in Berlin, Germany. Back home, he presented an update of his research at the annual meeting of the Association for Research in Vision and Ophthalmology in Sarasota, Florida and lectured on inherited eye diseases in the dog at the Cornell Symposium on Canine Health.

Gregory Acland and Bennett Hershfield presented the results of their research on canine progressive retinal atrophy at the annual meeting of the Association for Research in Vision and Ophthalmology in Sarasota, Florida.

Douglas Antczak presented the results of his studies on histocompatibility antigen expression in the placenta of the horse at the Sero Symposium on the Immunobiology of Reproduction in Boston. He also attended a Wellcome Trust meeting, "Immunological Reagents for the Study of Disease in Companion Animals" in London, where he reviewed the status of reagents for research and clinical application in horses. Many of the available reagents and techniques have been developed in his laboratory during the past 15 years.

Max Appel and Dina Barbis attended the 9th International Virology Congress in Glasgow, Scotland. Dr. Barbis's trip was underwritten by an NIH travel award from the American Society for Virology.

Judith Appleton was promoted this year to associate professor with tenure. Dr. Appleton, who was the 1991 winner of the SmithKline Beecham



At commencement ceremonies in October, the University of Göteborg, Sweden conferred the degree of Doctor Honoris Causa on Gustavo Aguirre, the Institute's Caspary Professor of Ophthalmology. The Faculty of Mathematics and Natural Sciences cited Dr. Aguirre's work on hereditary retinal degenerations and his progress toward the development of gene replacement therapy for the treatment of blindness associated with mucopolysaccharidosis, a severe and in some forms fatal inherited disease of dogs, cats, and humans.

Award for Research Excellence, came to the Institute as a postdoctoral associate in 1982. She was promoted to the rank of senior research associate in 1985 and assistant professor in 1987.

Dr. Appleton was invited to the University of Saskatchewan, Western College of Veterinary Medicine, where she gave two seminars describing her research.

Dina Barbis and Juli Maher have both been chosen by the NIH to receive National Research Service Awards. These very prestigious postdoctoral fellowships are awarded for a period of three years.

Robin Bell delivered two invited lectures at Louisiana State University College of Veterinary Medicine in Baton Rouge. He also chaired a session of the Eighth International Congress on Trichinellosis in Orvieto, Italy and was elected to the International Commission on Trichinellosis.

DEGREES CONFERRED

Theodore Llana, III: Ph.D.: "The Regulation of Intestinal Immunity Following *Trichinella Spiralis* Infection." Dr. Llana is attending business school at Georgetown University.

Laura Kay Hanson: Ph.D.: "Mechanisms and Functional Effects of Antigenic Variation in the Hemagglutinins of Equine Influenza Viruses." Dr. Hanson is now a postdoctoral associate in the Department of Microbiology and Immunology at Eastern Virginia Medical School.

Nancy Burton-Wurster gave a seminar on her work on osteoarthritis at Miles Laboratories in West Haven, Connecticut.

Leland Carmichael presented a series of lectures and seminars to veterinarians and scientists in Sollefteå and Uppsala, Sweden. He was invited to the University of Querataro, Mexico, where he gave a short course on canine infectious diseases to veterinary students and clinicians. During the September meeting of the Advisory Committee to the Dean of the Postgraduate Division of the School of Veterinary Medicine (UNAM), Mexico City, he was nominated to the Academia Veterinaria Mexicana.

Vicki Meyers-Wallen continues as Chief-of-Service of the Small Animal Fertility and Infertility Clinic at the Veterinary Teaching Hospital. She also serves on the national board examination committee of the American College of Theriogenologists and was

an invited speaker at the Canine Symposium that they sponsored jointly with the Society for Theriogenology.

Colin Parrish and Allen Gruenberg presented their studies of the antigenic structures of parvoviruses and their ability to infect dogs and cause disease at the annual meeting of the American Society for Virology in Davis, California. Four members of their laboratory took part in the Fifth International Parvovirus Workshop held in Crystal River, Florida. Dr. Parrish was invited to chair the session on the structure of parvovirus capsids and also discussed his investigations of the host ranges of parvoviruses.

Jharna Ray attended the 8th Annual Conference of the National Mucopolysaccharidosis Society in Philadelphia and presented the results of a study, "Beta-Glucuronidase Mutation in Retinal Pigment Epithelium Causing Mucopolysaccharidosis VII" at the annual meeting of the Association for Research in Vision and Ophthalmology in Sarasota, Florida.

Kunal Ray delivered a lecture, "Vitamin D-Binding Protein Gene: Structural Analysis and Functional and Evolutionary Correlations," at the National Eye Institute of the NIH.

Staff of the Baker Institute

ADMINISTRATION

Gustavo D. Aguirre

Director: V.M.D., Ph.D., U. of Pennsylvania

Susan Howell Hamlin

Administrative manager: B.S., Elmira College

Carlene M. Furch

Administrative assistant

Anita S. Hesser

Secretary to the director and assistant systems administrator

Dorothy K. Scorelle

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Sharon E. Morrow

Accounts assistant

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Robin Fisher Cisne

Staff writer: B.A., Ithaca College; M.A., SUNY Binghamton

Judith L. Halpin

Public affairs assistant

EMERITUS

Ben E. Sheffy

Caspari Professor of Nutrition, Emeritus: B.S., M.S., Ph.D., U. of Wisconsin

LABORATORIES

Giralda Laboratory for Canine Infectious Diseases

Leland E. Carmichael

John M. Olin Professor of Virology: A.B., D.V.M., U. of California; Ph.D., Cornell

Patricia L. Lucia

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David N. Peters

Graduate research assistant: D.V.M., Ohio State U.

Amy L. Kloster

Laboratory technician: B.S., William Smith College

Hadley C. Stephenson Laboratory for the Study of Canine Diseases

Max J. G. Appel

Professor of virology: Dr. med. vet., U. of Hannover; Ph.D., Cornell

Reinhard Straubinger

Postdoctoral fellow: D.V.M., U. of Munich

Mary Beth Matychak

Research technician: U. of Evansville

Bostwick Laboratory of Molecular Biology

Colin R. Parrish

Assistant professor of virology: B.Sc., Massey U.; Ph.D., Cornell

Allen Gruenberg

Postdoctoral associate: B.S., Massey U.; M.S., Ph.D., Monash U.

A. T. M. Wahid

Postdoctoral associate: M.D., Chittagong (Bangladesh) Medical College

Dina P. Barbis

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Laurel E. Southard

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Wendy S. Weichert

Laboratory technician: B.S., Cornell

John M. Olin Laboratory for the Study of Canine Bone and Joint Diseases

George Lust

Professor of physiological chemistry: B.S., U. of Massachusetts; Ph.D., Cornell

Nancy Burton-Wurster

Senior research associate: B.A., M.S., Ph.D., New York U.

Anthony R. C. Farquhar

Postdoctoral associate: B.S., U. of Massachusetts; M.S., Ph.D., Cornell

Dai-Wei Zhang

Graduate research assistant: M.D., Beijing Medical U.

Elizabeth Grisanzio

Laboratory technician: B.S., U. of Vermont

Margaret S. Vernier-Singer

Laboratory technician: B.S., Ohio State U.

Alma J. Williams

Laboratory technician: B.A., U. of Pennsylvania; M.S., Cornell; AALAS accreditation

Laboratory of Cellular Growth and Differentiation

James N. MacLeod

Assistant professor of molecular genetics: B.S., U. of Delaware; V.M.D., Ph.D., U. of Pennsylvania

Da-Nian Gu

Postdoctoral associate: B.S., M.S., Ph.D., Fu Dan U., Shanghai, PR China

Immunology Laboratory

Robin G. Bell

Professor of immunology: B.Sc., Australian National U.; Ph.D., John Curtin School of Medical Research

Theodore Llana III

Graduate research assistant: B.S., U. of Maryland

Deborah Negrao-Correa

Graduate research assistant: B.S., U. Estadual de Campinas; M.S., U. Estadual de São Paulo

Clarissa L. Santos

Graduate research assistant: B.S., U. of Philippines

Lincoln S. Adams

Research technician: B.S., Hobart College; AALAS accreditation

Mucosal Immunity Laboratory

Judith A. Appleton

Associate professor of immunology: B.S., Indiana U.; M.S., Ph.D., U. of Georgia

Prema Arasukavalar

Postdoctoral associate: B.S., National U. of Malaysia; M.S., U. of Wyoming; Ph.D., Hahnemann U.

Lauri A. Ellis

Graduate research assistant: B.S., Pennsylvania State U.

Laura K. Hanson

Graduate research assistant: B.S., U. of Washington

Lucille F. Gagliardo

Laboratory technician: B.S., Southampton College

Jeb B. Oblak

Laboratory technician: B.A., Ithaca College



Clockwise from left:
Sue Hamlin, administrative manager and Gabi Grünig, graduate research assistant; administrative staff members Judy Halpin, Dorothy Scorelle, and Carlene Furch

Equine Genetics Center

Douglas F. Antczak

Dorothy Havemeyer McConville Professor of Equine Medicine: B.A., Cornell; V.M.D., U. of Pennsylvania; Ph.D., Cambridge U.

Gabriele H. Grünig

Graduate research assistant: Dr. med. vet., U. of Zurich

Juli K. Maher

Graduate research assistant: B.S., D.V.M., U. of Illinois

Jessica M. Baker

Laboratory technician: B.A., U. of Vermont

Sarah E. Deacon

Laboratory technician: B.A., Smith College

Jane M. Miller

Research aide: B.S., M.P.S., Cornell

Laboratory for the Study of Inherited Canine Reproductive Diseases

Vicki N. Meyers-Wallen

Assistant professor of reproduction: B.S., U. of Maryland; V.M.D., Ph.D., U. of Pennsylvania; Diplomate, American College of Theriogenologists

Vicky L. Palmer

Laboratory technician: B.S., Houghton College





Public Affairs staff members Jeanne Truelsen and Robin Cisne

PASSAGES

Accounts Coordinator **Nancy Combs** retired from the Institute in December after completing 20 years of service at Cornell. Before joining the staff of the Institute in 1978, Mrs. Combs worked as an accounting clerk in the Entomology Department. Her son Ray is an animal technician at the Institute.

Charles B. Bailor, Sr., a retired animal technician, died June 4 at age 64. Mr. Bailor was a founding member of the Institute staff who served Cornell University with singular dedication for 36 years. A lifelong resident of the Ithaca area, he is survived by seven children, 15 grandchildren, two great grandchildren, and many friends.

Donnelley Laboratory of Gene Regulation and Expression

Jharna Ray

Assistant professor of molecular genetics: B.S., M.S., Ph.D., U. of Calcutta

Yanggeng Wu

Research associate: M.D., Beijing Medical U.

Inherited Eye Disease Studies Unit

Gustavo D. Aguirre

Alfred H. Caspary Professor of Ophthalmology

Gregory M. Acland

Senior research associate: B.V.Sc., U. of Sydney

Bennett G. Hershfield

Senior research associate: B.S., Jersey City State College; Ph.D., U. of Chicago

Kunal Ray

Senior research associate: B.S., M.S., Ph.D., U. of Calcutta

Kathryn E. Gropp

Research associate: S.B., Massachusetts Institute of Technology; D.V.M., Ph.D., U. of Florida

Victoria J. Baldwin

Laboratory technician: B.A., Colby College

Susan E. Pearce-Kelling

Laboratory technician: B.S., M.S., Cornell

COMPUTERS AND NETWORKS

Paul J. Lutwak

Systems analyst: B.A., B.S., Miami U.

ANIMAL CARE

Roy L. Barriere

Vivarium supervisor: AALAS accreditation

Raymond M. Combs

Animal technician: AALAS accreditation

Raymond J. Corey

Animal technician: A.A.S., SUNY Agricultural and Technical College, Delhi; AALAS accreditation

Kevin T. Draiss

Animal technician: A.A.S., SUNY Agricultural and Technical College, Delhi; B.S., Cornell, AALAS accreditation

James C. Hardy

Research aide: B.S., Cornell; AALAS accreditation

Laura A. Michel

Animal technician: A.A.S., SUNY Agricultural and Technical College, Farmingdale; B.S., Cornell; AALAS accreditation

Bruce W. Pastrick

Animal technician

Rita H. Sims

Animal technician: AALAS accreditation

Larry S. Wager

Animal technician

MAINTENANCE

Arthur D. Howser

Facilities Coordinator

Jeannette R. Carney

Laboratory attendant

Kathleen L. Crispell

Custodian

Ralph L. Crispell

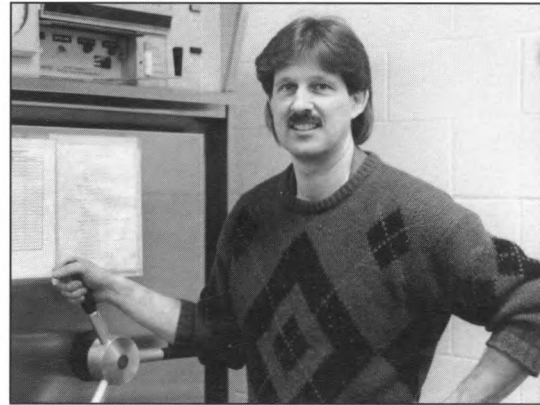
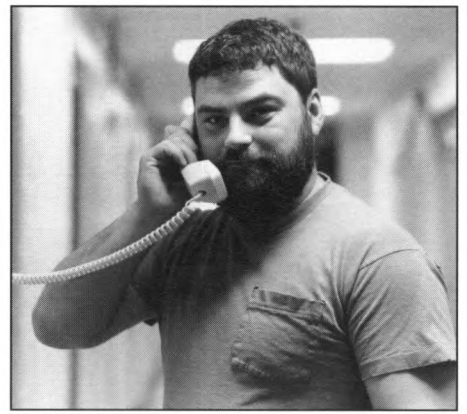
Custodian

Richard E. Daniels

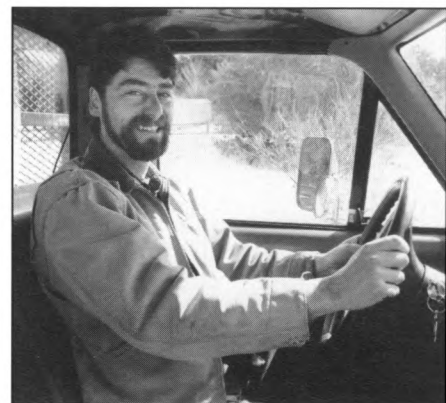
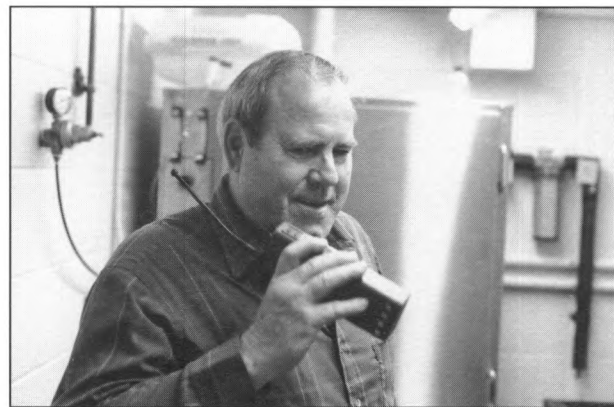
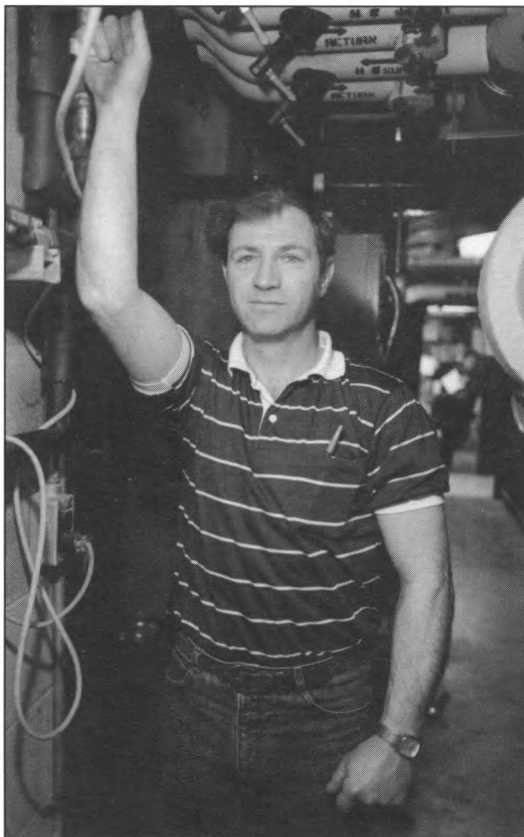
Maintenance mechanic

Russell F. Haus

Maintenance mechanic



Animal care staff members (clockwise from left), Ray Corey and Larry Wager, Ray Combs, and Kevin Draiss



Maintenance staff members (clockwise from above), Russ Haus, Art Howser, Rick Daniels, and Jean Carney

Giralda Laboratory for Canine Infectious Diseases

MOST CAUSES OF DEATH in newborn puppies are poorly understood by veterinary clinicians and breeders, and it has long been a goal of our research team to identify the infectious agents responsible for neonatal mortality in dogs. Our current work targets viruses that affect the canine reproductive tract, fetuses, and pups less than one month of age.

Minute virus of canines (MVC) has been known for over 20 years and was believed to be nonpathogenic, but we have recently discovered that it can cause sickness and even death in puppies. The inconsistent and unpredictable course of this infection makes it difficult to diagnose and prevent: of the MVC-infected

puppies we have seen, most showed only mild respiratory symptoms or diarrhea, but the disease can cause serious breathing difficulties and enteritis in some pups and has been responsible for embryo resorption and fetal death in other cases.

The severity of a puppy's response to infection with MVC can vary a great deal depending on a combination of factors, including the intensity of exposure to the virus, the virulence of the viral

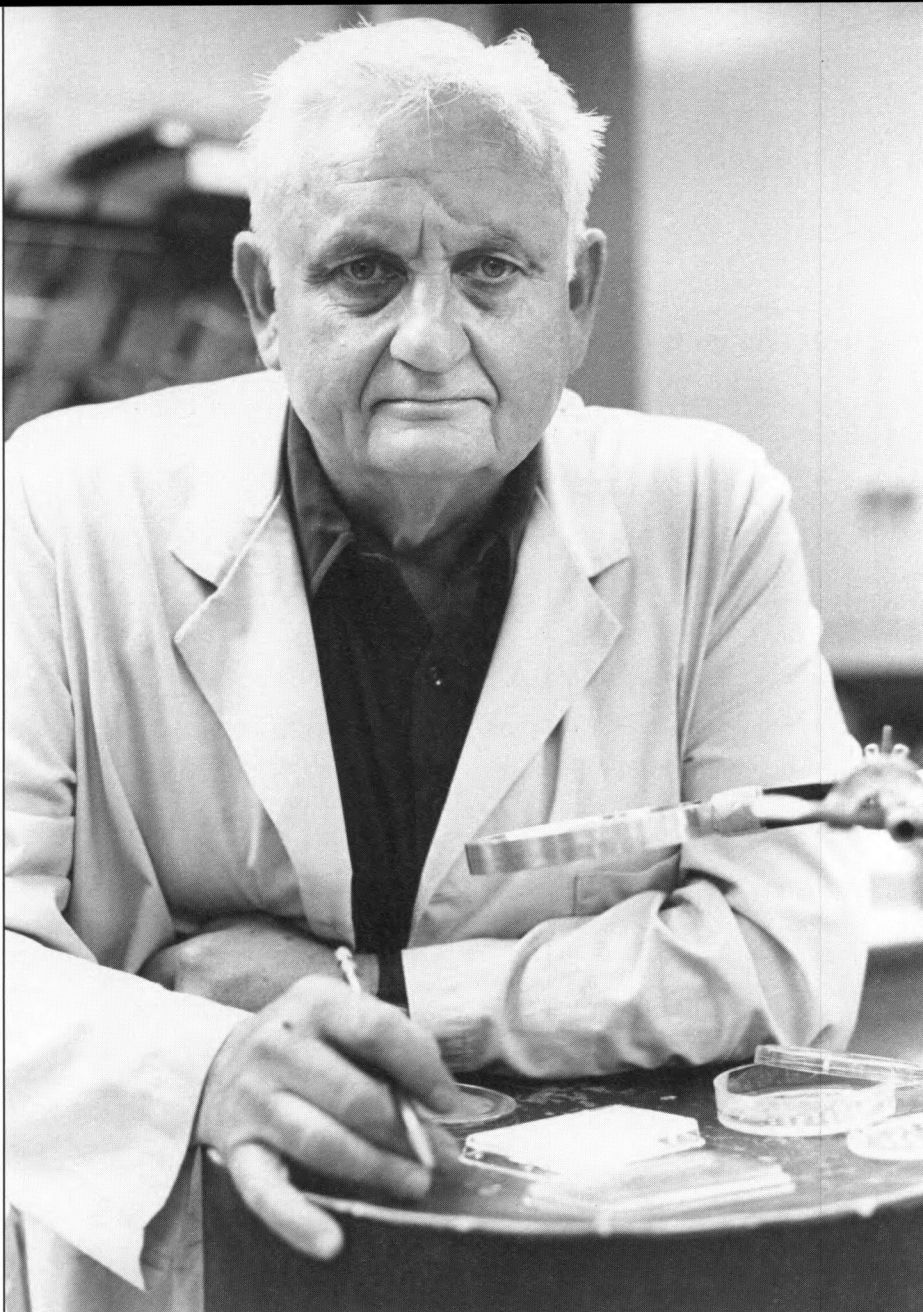
strain, and the possibly heightened susceptibility of purebred dogs to infection. The disease signs in fatal field cases,

other than "sudden death," have consisted of difficulty in breathing, crying, vomiting, and diarrhea. In some instances, pups were stillborn with body edema and damage to the heart. Unlike the field cases, experimental infections resulted primarily in respiratory disease, with little obvious damage to the intestinal tract. Reasons for the marked discrepancy between natural and experimental cases are not known.

We have been successful in developing rapid and specific diagnostic criteria for MVC, but there is a need for wider recognition of the disease by dog breeders and veterinarians. The virus is widely prevalent in the dog population, for recent serological tests of over a thousand samples from breeding females sent to the New York State Diagnostic Laboratory indicate an infection rate of at least 50 percent. With support from the American Kennel Club and donations from other kennel clubs, we have launched an effort to amass case histories and obtain specimens from pups suspected of dying of MVC infection in order to determine the significance of the disease to the general canine population.

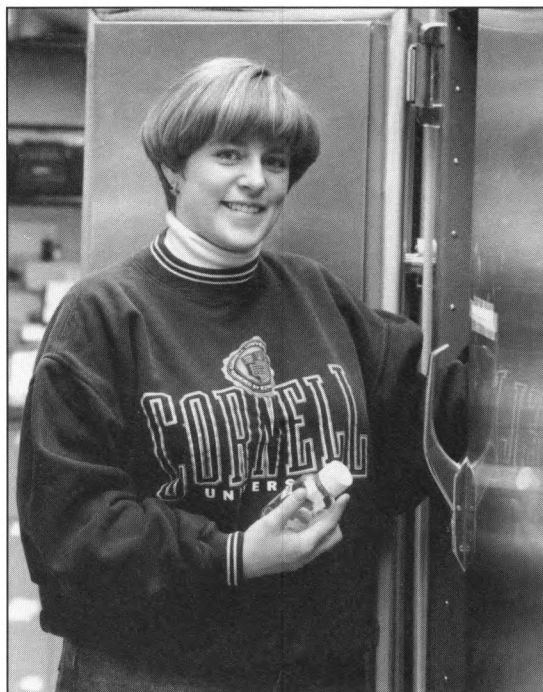
If MVC is found to be a major cause of illness and death in neonatal dogs, a vac-

Minute virus of canines is widely prevalent in the dog population. If it is found to be a major cause of illness and death in neonatal dogs, a vaccine could be developed quickly.



cine could be developed quickly. An experimental vaccine has already been tested that protects fetal and neonatal pups against the virus, but before MVC vaccination can be considered routine, we must determine whether the risk is severe enough to warrant yet another vaccine. Over the next year, we hope to acquire enough data to enable us to make a recommendation on the appropriateness of MVC vaccination for dogs.

—Leland E. Carmichael



Clockwise from top left:
*Skip Carmichael, Dave
Peters, and Amy Kloster*

Hadley C. Stephenson

Laboratory for the Study of Canine Diseases

OUR RESEARCH EFFORTS during the past year have concentrated on the vaccination and immunization of dogs against Lyme disease. A killed vaccine containing whole organisms has been available for use in dogs since 1990, but there are concerns about its efficacy and safety.

We are using a new approach to develop a vaccine from a single *Borrelia burgdorferi* protein that appears to provide more reliable protection against Lyme disease and the chronic polyarthritis that most dogs develop after tick exposure.

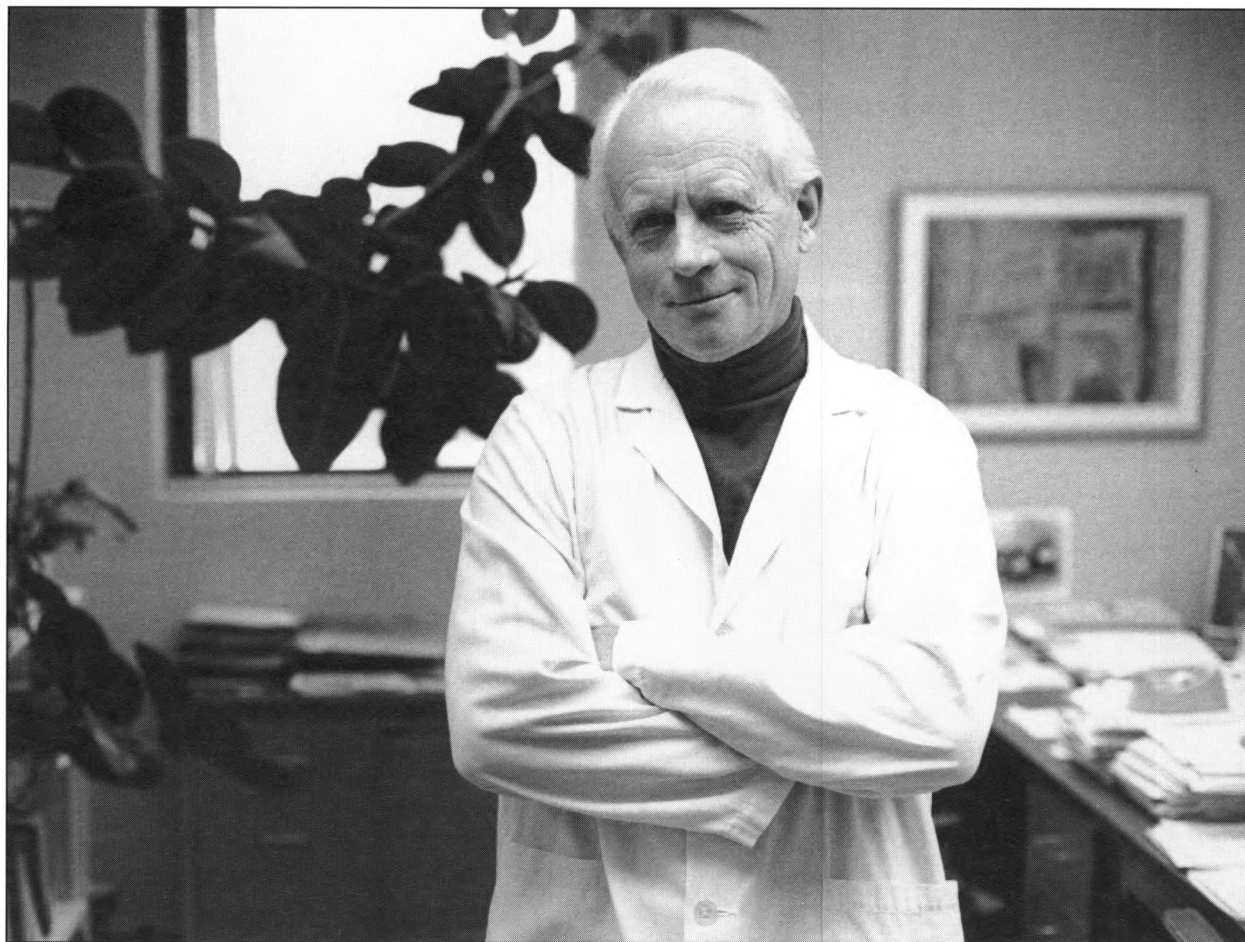
During the last year, we found that outer surface protein A (Osp A) of *B. burgdorferi*, the spirochete that causes Lyme disease, is a good candidate for canine as well as for human vaccines, and our preliminary data from Osp A trials confirm this. Dogs were protected not only from disease but also from infection when challenged with *B. burgdorferi*-infected ticks.

*We have found
that outer surface
protein A is a good
candidate for canine
as well as human
vaccines.*

The *B. burgdorferi* Osp A protein that we used for the vaccination trial is a recombinant product engineered in the common bacterium *E. coli* by Yung-Fu Chang, one of our colleagues in the Veterinary Diagnostic Laboratory. Dogs were vaccinated twice with Osp A and then exposed to *B. burgdorferi*-infected ticks. Three to four months after tick exposure, immunized dogs were

completely free of spirochetes, an indication that the vaccine was effective. Our recent studies also found that dogs that had been vaccinated with Osp A did not develop arthritis. We have concluded that immunizing dogs with Osp A protein protects them from Lyme infection and disease when they are exposed to *B. burgdorferi*-infected ticks. However, these data are still preliminary, and we must confirm our results with more study.

Work done this year by Richard Jacobson, another of our collaborators in the Veterinary Diagnostic Laboratory, highlighted some of the concerns about the present commercial vaccine. He surveyed serum samples from dogs in the field using a special test that makes it possible to distinguish among dogs that have been vaccinated, dogs that have been exposed to tick infection, and dogs that have both natural and vaccine-induced antibodies to *B. burgdorferi*. In sampling dogs from endemic areas that had been clinically diagnosed with Lyme disease, Dr. Jacobson discovered that about one third showed evidence of vaccination, but not of tick exposure. Another 25 percent of the dogs tested had evidence of both vaccination and tick exposure. This result implies that the



vaccine either did not protect those dogs from natural infection or did not defend against the effects of prior infection, as the product insert suggested it would.

A highlight this year was the arrival of Reinhard Straubinger, a recent graduate of the College of Veterinary Medicine of the University of Munich in Germany. Reinhard spent the summer of 1992 as a student in our laboratory and has returned to investigate the pathogenesis of Lyme arthritis as a postdoctoral fellow. His work is being supported by a grant from the Mercedes-Benz Company.

— *Max J. G. Appel*



Max Appel and Mary Beth Matychak

Albert C. Bostwick

Laboratory of Molecular Biology

HOW DO VIRUSES INFECT ANIMALS? What factors limit their ability to infect a new species? What determines the ability of a virus to cause disease? These are the questions that we are examining in our studies of canine parvovirus (CPV) and the related feline panleukopenia virus (FPV).

The parvovirus that emerged in 1978 as a new disease of dogs appears to have been a variant of FPV. The mutation consisted of a few small additions in the amino acid residues on the outer surface of the feline virus particle. To determine how those changes enabled it to replicate efficiently and cause disease in a new species, we are focusing on the critical but

poorly understood process by which a virus enters a host cell, and the virus protein coat and viral DNA genome separate. So far, our data indicate that a virus attaches to the outer membrane of the cell and is taken up into vesicles, bubbles formed of membrane that float in the cell's cytoplasm. At some point, the virus or

the viral DNA must be able to leave the vesicles and enter the cytoplasm; we believe that by discovering the mechanism involved in this step we will understand how the differences we have identified between the canine and feline viruses determine their ability to infect their respective host species. The next

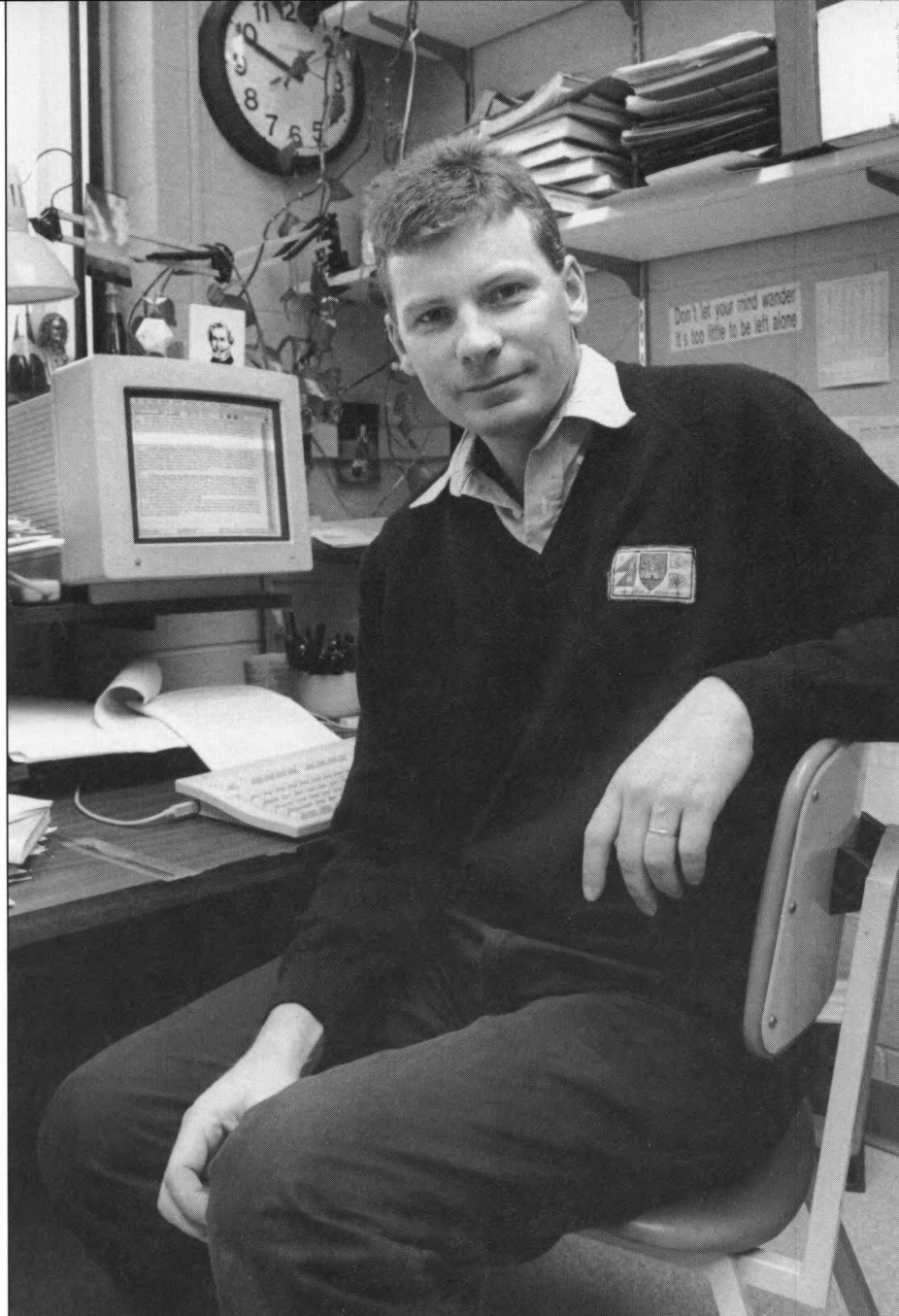
step is known: once viral DNA reaches the cytoplasm, it can travel to the cell nucleus, where it replicates.

VACCINE DEVELOPMENT

CPV strains have been changing in nature since the disease first emerged. The original strain, CPV-2, has rarely been found in nature since about 1981, but it is still used in most modified-live vaccines. Although those vaccines still protect against CPV-2b, the antigenic variant of CPV that is currently infecting dogs, we have been developing a new, homologous vaccine in collaboration with Leland Carmichael. The candidate vaccine strain we have derived from CPV-2b appears to be safe and effective, and we are initiating field trials to determine its efficacy as a vaccine for dogs in the pet population.

As part of that study, Allen Gruenberg cloned the DNA from the attenuated strain into a bacterial plasmid and examined its base sequence in detail. Dr. Gruenberg has shown that only a few nucleotide changes distinguish it from the virulent strain from which it is derived. It is probable that those changes slow the replication of the attenuated virus in the body enough to allow dogs to develop an immune response and elimi-

We are developing a new vaccine derived from CPV-2b, the parvovirus strain that is currently infecting dogs.



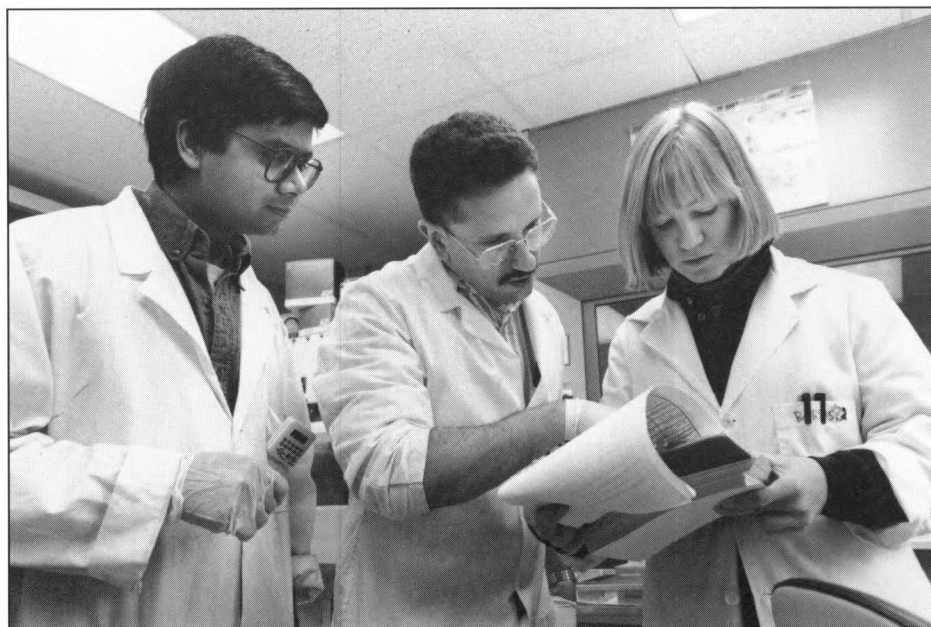
Left: Colin Parrish; Below: Laurel Southard and Dina Barbis; Bottom: A. T. M. Wahid, Allen Gruenberg, and Wendy Weichert



nate the virus before it can take hold in the intestine and cause clinical disease.

Our work this year has incorporated a number of new technologies, particularly in the analysis of protein structures. We have acquired a Silicon Graphics computer capable of displaying the three-dimensional structure of proteins. By examining in detail the ways in which the virus proteins are folded, we can increase our understanding of their functions.

—Colin R. Parrish



The John M. Olin Laboratory for the Study of Canine Bone and Joint Diseases



HIS YEAR MARKED THE 25TH ANNIVERSARY of our laboratory and our efforts to identify the causes and means of controlling hip dysplasia and osteoarthritis. This has proven to be a very difficult area of study for researchers here and elsewhere, but we continue to make progress in our efforts to pinpoint the factors that predispose a puppy to hip dysplasia and to characterize the biochemical changes underlying the progression from hip dysplasia to osteoarthritis.

HIP DYSPLASIA

During the past few years, research conducted here and by our colleagues at other laboratories has conclusively identified several factors that increase a dog's

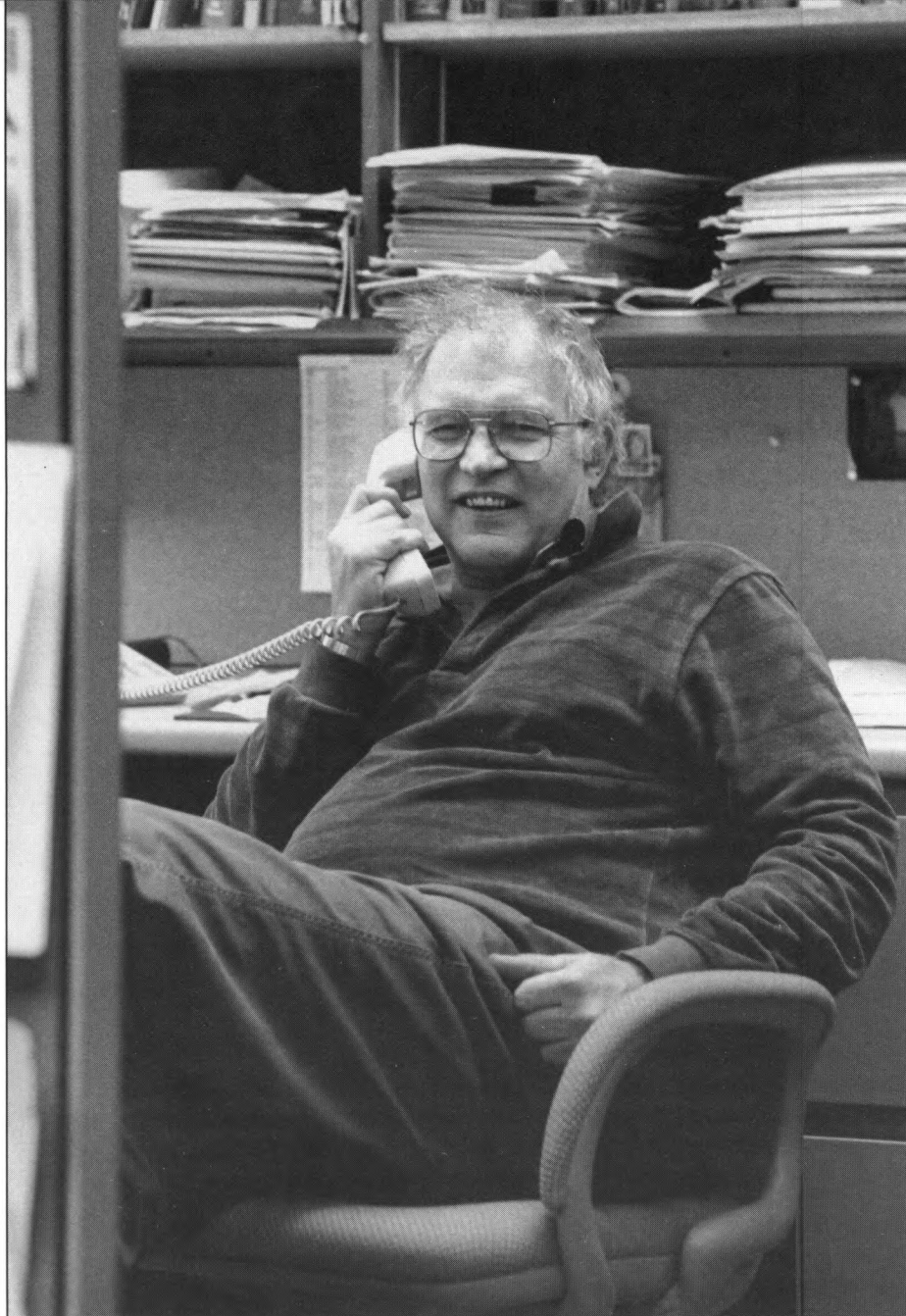
risk of developing hip dysplasia. Foremost among them is high food consumption early in life. Optimum nutrition promotes rapid growth, which in turn increases the likelihood that hip dysplasia will develop. Another important factor is the degree of laxity in a puppy's hip joints: joints that can easily be displaced more than four millimeters are more likely to become

dysplastic than tighter joints. These observations were confirmed by studies that demonstrated that limiting puppies' calorie intake and treating them with Adequan®, a drug that protects cartilage from deterioration, during the rapid growth period from two to eight months

of age prevented hip dysplasia from developing. However, while these studies provide guidelines for prevention and treatment, they offer no explanation of the primary cause of the disease in dogs.

Some of our most recent findings have implied that exposure to high levels of the hormone relaxin before and after birth may contribute to the development of hip dysplasia. We observed that dysplastic bitches have prolonged high levels of relaxin in their blood and milk after gestation, and that pups nursing on these bitches have the hormone in their blood. Produced during the latter third of pregnancy, relaxin loosens the pelvic opening to facilitate delivery, and we postulated that the effect of relaxin on the hip joints of puppies is similar to routine gestational changes. In collaboration with our colleague Bernard Steinetz of New York University, we have devised a study that will help determine the role of this hormone in the disease. We are preparing polyclonal and monoclonal antibodies to purified dog relaxin in order to learn whether injection of genetically susceptible pups with the relaxin antibodies prevents the development of hip dyspla-

*We want to learn
whether injection of
genetically susceptible
pups with relaxin
antibodies prevents
the development of
hip dysplasia.*



*George Lust and
Alma Williams*

CHARLES HARRINGTON



sia. The discovery of a reliable method to protect predisposed pups from hip dysplasia will then make it possible for us to infer the mechanism of the disease.

OSTEOARTHRITIS

Normal articular cartilage is a smooth, resilient tissue that minimizes friction from joint movement and resists the compression caused by putting weight on a joint. The osteoarthritis associated with canine hip dysplasia may be a consequence of damage to cartilage that results from the abnormal distribution of forces in a misaligned joint. We have been

studying the response of articular cartilage to various mechanical factors in order to learn how they may influence the development of osteoarthritis.

The constitution of cartilage includes proteoglycans, large molecules that tend to absorb water and swell. This tendency is counterbalanced by the confinement of proteoglycans within a collagen fibril network. We hypothesize that subtle disruption of the collagen fibril network or damage to the cartilage cells results in the appearance of more obvious osteo-

arthritic changes. All osteoarthritic cartilage shows a loss of proteoglycan, although we also found that it contains elevated levels of other components, including fibronectin, an important cell adhesion glycoprotein, early in the course of the disease. The effects of these constituents and others on the supra-molecular organization of cartilage or the behavior of cartilage cells are not well understood.

To identify the critical biochemical or biomechanical events that cause osteo-

arthritis, we are attempting to simulate in tissue culture the conditions under which articular cartilage is maintained in a living, working joint. We think that repetitive mechanical stresses equivalent to moderate daily activity may be essential to maintain cartilage in a healthy state. Tony

Farquhar, a postdoctoral


associate in our laboratory, has drawn on his training in mechanical engineering to develop a bio-material testing system that can apply either moderate or heavy dynamic loads of up to nine times body weight to a sample of cultured cartilage. By subjecting cartilage explants to heavy dynamic loads, Dr. Farquhar hopes to induce some of the early biochemical changes that we have observed in osteo-

arthritic tissue. Such an *in vitro* model of osteoarthritis would permit potentially useful therapeutic agents to be screened quickly and efficiently for drug testing and development.

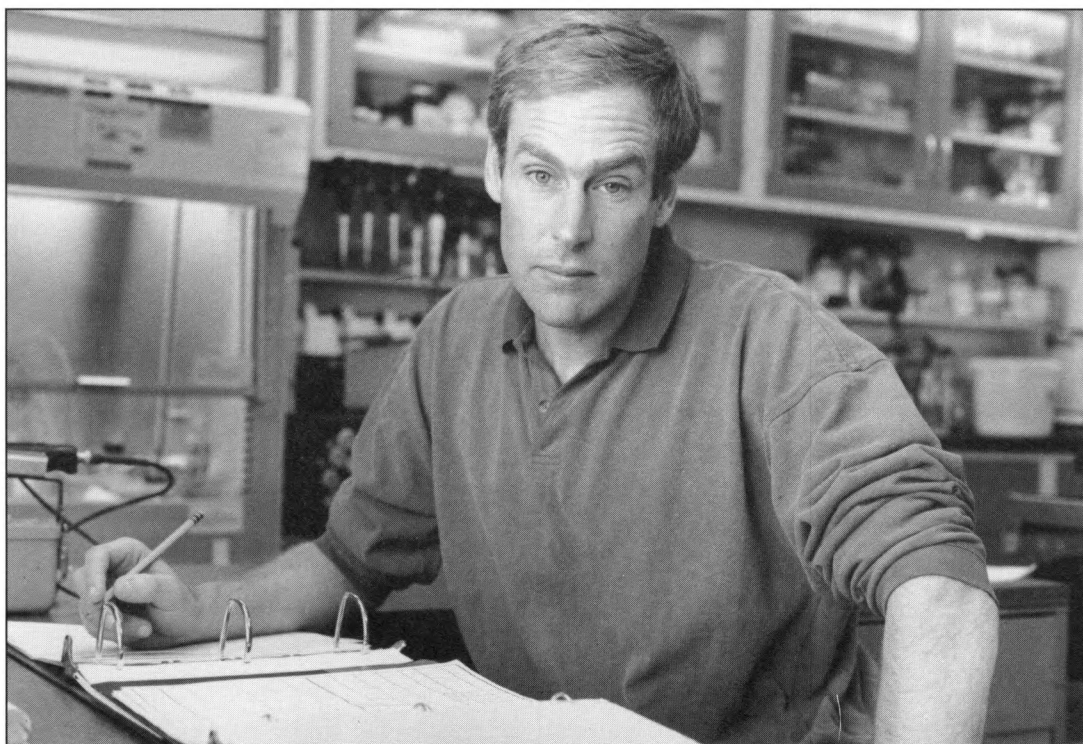
Since beginning this testing last year, Dr. Farquhar and Margaret Vernier-Singer have determined that cartilage explants synthesize additional proteoglycan and protein in response to moderate cyclical loads, while heavier loads inhibit those processes. They have also found that the response of the cartilage to load varies according to the area of the joint from which it was taken. For example, preliminary results suggest that explants from joint areas that would normally be subjected to high loads were more resistant to the effects of high loads in culture.

The kinds of studies that we have done so far require destruction of the explant to gain biochemical information. It would be very useful if we could instead follow the same explant through time or a series of treatment. Dr. Farquhar is continuing collaborations with John Bertram of the Department of Anatomy to develop new methods of mechanical assessment of articular cartilage. We are also working with Lynn Jelinski and Yang-Xia of Cornell's Biotechnology Program to apply quantitative magnetic resonance imaging (MRI) to articular cartilage. Eventual transfer of non-invasive methods such as MRI from laboratory explants to clinical care could be of great benefit to dogs and other species that suffer from osteoarthritis.

—George Lust and
Nancy Burton-Wurster



We are attempting to simulate in culture the conditions under which cartilage is maintained in a living, working joint.



*Dai-Wei Zhang, Nancy
Burton-Wurster (above),
and Tony Farquhar*

Laboratory of Cellular Growth and Differentiation



OUR STUDIES IN THE PAST YEAR continued and expanded our research into the mechanisms of bone growth, joint motion, and the cellular production of cartilage.

We hope that a fuller understanding of the molecular interactions that contribute to abnormal bone growth and degenerative arthritis will enable the development of improved methods to prevent and treat these human and animal diseases.

BONE GROWTH: THE TALL AND THE SHORT OF IT

Bones grow through the proliferation and maturation of cells called chondrocytes, which produce cartilage that is subse-

quently mineralized to form new bone tissue.

Chondrocytes are found in the growth plates located at the ends of long bones. The rate and extent of bone growth is largely influenced by genetic factors, but hormones, the environment, and disease are also influential. We do not yet understand the molecular events regulated in chondrocytes that govern new bone formation, and we are trying to

identify the genetic controls on that process.

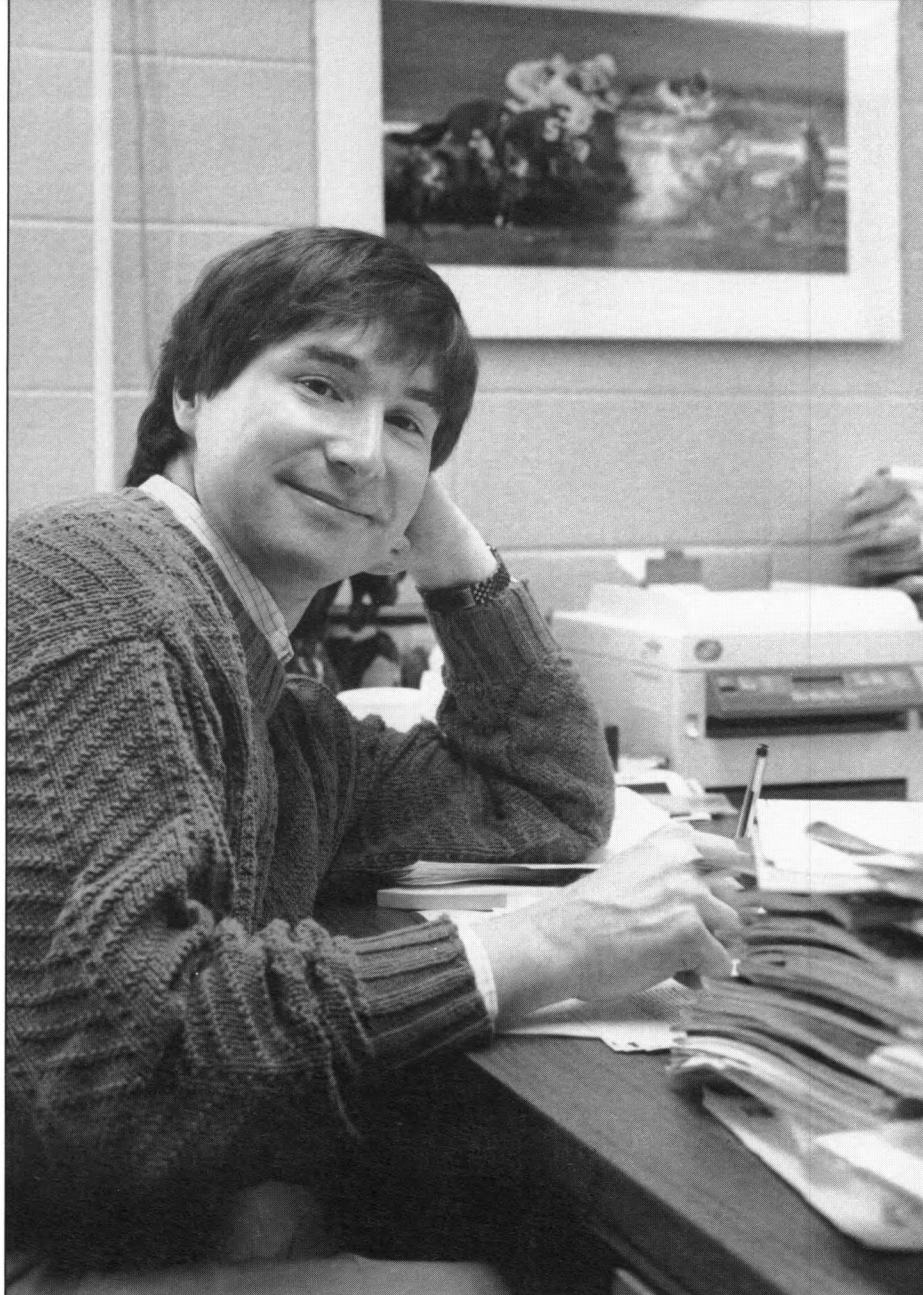
We hope to understand chondrocyte proliferation and maturation through study of chondrodysplasia, an inherited

form of dwarfism. Although considered the standard in some dog breeds, such as Dachshunds and Basset Hounds, this condition is recognized as a disease in other dog breeds, as it is in humans. We are working with mice to identify the genes responsible for the condition and to define the mechanism by which chondrodysplasia and related diseases alter the proliferation, differentiation, maturation, and degeneration of chondrocytes in growth plates. Da-Nian Gu is working on the isolation of gene sequences and the preparation of DNA probes that are critical to chondrocyte research.

ARTHRITIS: THE TROUBLE WITH JOINT CARTILAGE

Cartilage is also found on joint surfaces, covering the bone ends to provide protection and allow smooth movement. In contrast to their growth plate counterparts, chondrocytes in articular (joint) cartilage are long-lived and rarely divide. They maintain stability in the molecular components of articular cartilage responsible for important biomechanical properties such as strength, durability, and deformability. In arthritic diseases, these molecules are degraded, causing painful joint inflammation and difficult movement. A major problem in the treatment of arthritis is that articular cartilage has very poor regenerative potential. Most current therapies are designed for symptomatic pain relief and the preven-

If articular cartilage cells can be stimulated to divide and re-synthesize normal cartilage, it may be possible to restore function to arthritic joints.



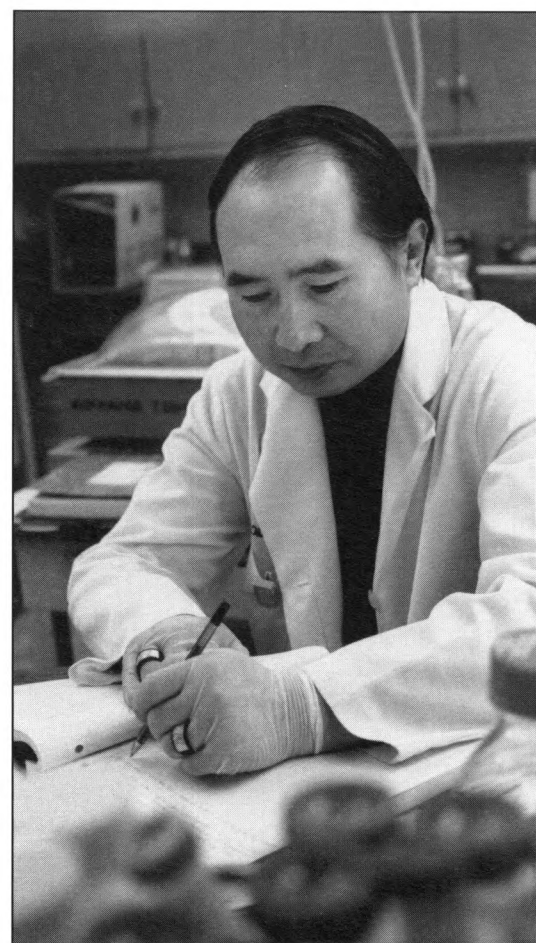
tion of further degradation, not to rebuild normal cartilage. We believe that if articular chondrocytes can be stimulated to divide and re-synthesize normal cartilage, it may be possible to restore function to arthritic joints.

Our approach to this exciting possibility is two-pronged. First, by studying changes in chondrocyte gene expression that occur during arthritic degeneration, we hope to understand the disease on the molecular level. Second, by comparing gene expression in the rapidly dividing chondrocytes of the growth plate to the non-dividing chondrocytes that make articular cartilage, we hope to identify the

genes that regulate chondrocyte division and growth. We have initiated work in both areas.

In the summer, we were joined by two veterinary students on ten-week research fellowships: Linda Abraham of the Royal Veterinary College in England, who was supported by the Dorothy Russell Havemeyer Foundation; and Gita Nagassar-Mohit of Tuskegee University, who was assisted by the Woodruff Foundation. Both of these students were excellent, and I am fortunate to have had the opportunity to work with them.

—James N. MacLeod



*Jamie MacLeod (left) and
Da-Nian Gu*

FOR SEVERAL YEARS we have been examining the biological properties of IgE, an antibody best known for causing allergic reactions. We believe that an exact understanding of how IgE functions will yield better treatments for allergies such as flea dermatitis or hay fever and asthma.

We are also interested, however, in exploring our finding several years ago that, in the case of infection with *Trichinella spiralis*, IgE plays a protective role in the body. By defining more precisely how IgE operates in rejecting parasites, we also hope to improve our understanding of how IgE-mediated allergic reactions lead to unwanted pathology and disease.

We want to define more precisely how IgE, an antibody best known for causing allergic reactions, operates in rejecting Trichinella spiralis from the intestine.

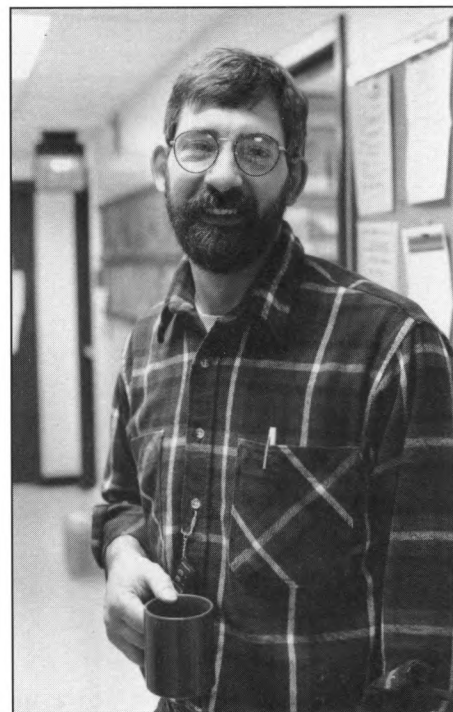
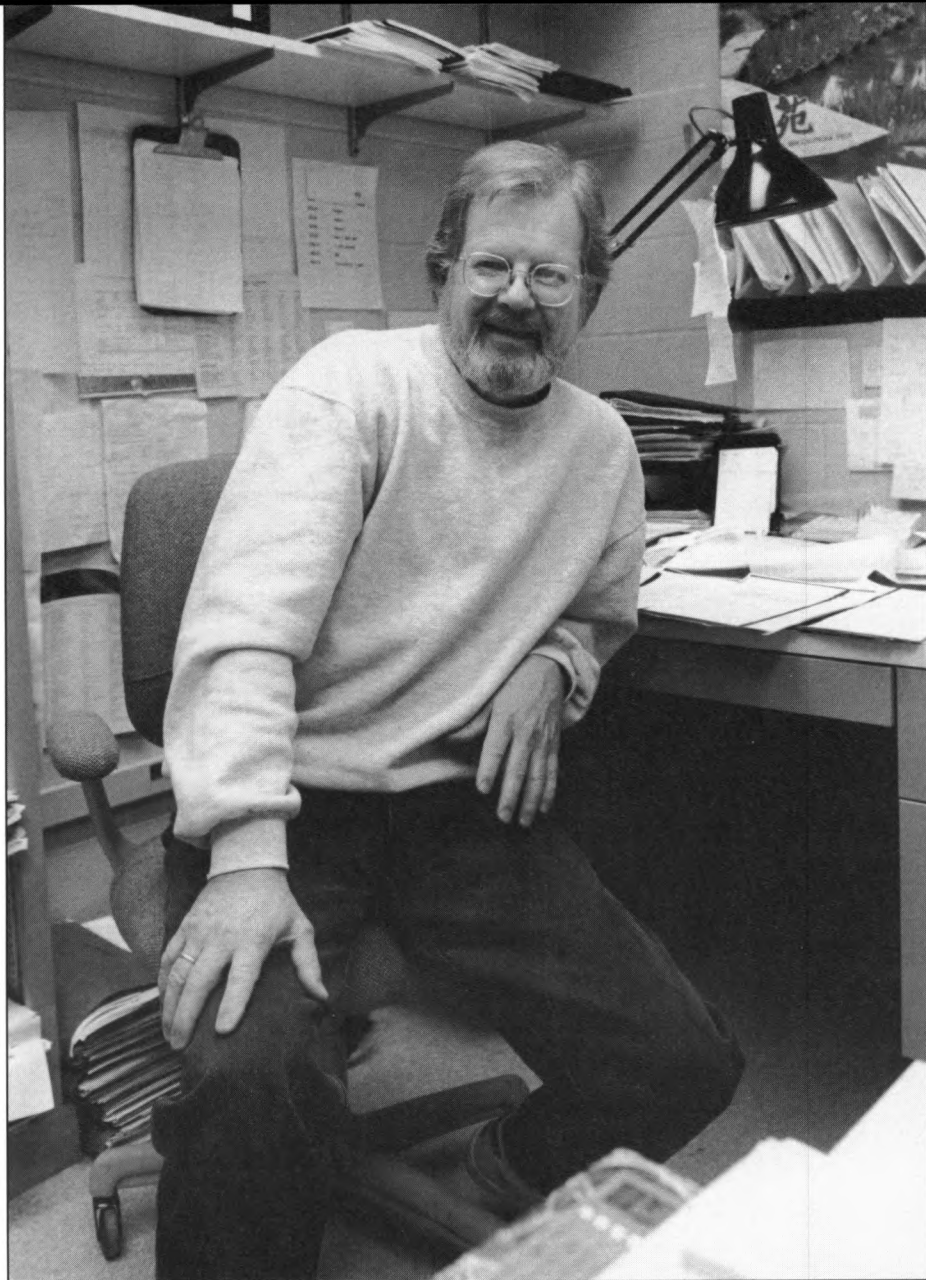
In previous work we demonstrated that transfusing *T. spiralis*-infected rats with parasite-specific immune cells, or lymphocytes, and then with purified, specific IgE caused the elimination of the parasite from the intestine, where it resides. We observed a pronounced uptake of IgE in the intestine, but not elsewhere in the body, indicating that intestine-specific binding of IgE was occurring. Our

experiments with uninfected rats, which produce no activated immune cells, indicated that the presence of the lymphocytes—or a factor they release—appears to be required for the IgE antibodies to enter the intestine and initiate rejection of the parasites. These immune cells

“home” to the intestine in large numbers and appear to exert a predominately local effect in the intestine.

Our studies this year have shown that IgE is not carried to the gut through the liver by bile as are some other antibodies. We found that the bile transported only degraded IgE of low molecular weight, while we were able to recover biochemically intact, high-molecular-weight IgE from the intestinal lumen of bile-free rats. Our evidence suggests a transport mechanism that moves IgE from the rats’ bloodstream to the intestinal wall, where it can bind both to lymphocyte populations in the epithelium and to the cells that absorb nutrients.

As we observed earlier, IgE cannot enter the gut unless immune lymphocytes are also present. These cells secrete a variety of potent hormones, called cytokines, that stimulate growth and gene activation for a variety of cell types. One of these cytokines, IL-4, induces IgE production. Through a collaboration with Andrew McKnight of Oxford University, we acquired recombinant IL-4, injected it (rather than whole cells) into uninfected rats, and then measured IgE antibody uptake in the gut. The response was dramatic: we observed increases in IgE uptake comparable to those we had observed in infected rats or in uninfected rats injected with immune lymphocytes.



This is the first evidence that any cytokine can regulate IgE uptake and transport processes in the intestine.

Current studies are directed at measuring IgE uptake and transport mechanisms. We wish to know how much IgE is transported and how this relates to the levels that are observed in serum. It is possible that the intestine's IgE transport process helps keep serum levels of IgE down, thus preventing allergic reactions in the skin and upper respiratory tract. We also plan experiments to analyze the mechanism by which IgE acts to produce parasite rejection.

—Robin G. Bell



Clockwise from top left:
*Robin Bell; Linc Adams;
Deborah Negrao-Correa
and Clarissa Santos*

Mucosal Immunity Laboratory



OUR WORK THIS YEAR has continued our investigations into the regulation of antibody function in parasitized animals. For these studies, we have used rats infected with the nematode

Trichinella spiralis to model the mechanisms of intestinal mucosal immunity that are common to many other animal species.

A better understanding of how immune systems work to fight infection could have profound implications for both animal and human health.

To deepen our understanding of antibody production and functioning, we must evaluate increasingly subtle interactions. We are currently focusing on certain parasitic protein molecules that

To deepen our understanding of antibody production and functioning, we must evaluate increasingly subtle interactions.

simultaneously serve two critical, and opposing, purposes. These proteins are found on the surface of the parasite and in material secreted by it. We believe that the parasite relies upon these molecules to invade the host animal, although we do not yet understand how that mechanism operates.

These protein molecules also induce antibody production, and rejection of parasites begins when host antibodies bind to them. Although these operations seem contradictory, the host animal's antibody response is essential

for the parasite as well; the host species must survive for the parasitic species to prosper.

Unfortunately, these key proteins are found in vanishingly small quantities, making them difficult to study. Prema Arasu has been cloning the genes that code for the proteins so that we can produce them in large amounts. By studying the proteins and their genes, we hope to learn how they assist parasitic invasion and determine which proteins are essential to inducing an antibody response.

In a related investigation, Lauri Ellis discovered that the molecules Dr. Arasu has been studying are not simple proteins. When the parasite makes these proteins, it "decorates" them with sugar molecules. These decorations are chains (sometimes branched chains) of sugars called glycans. Ms. Ellis found that the antibodies that resist infection with *T. spiralis* bind not to the proteins, but to the glycans attached to the proteins. We do not fully understand the significance of this finding, but we have observed that many parasite proteins bear these glycans and that the glycans stimulate a very powerful antibody response against the proteins that bear them.

— Judith A. Appleton



Judy Appleton (left) and Lucy Gagliardo



STUDENT WORK RECOGNIZED

Prema Arasu and **Lauri Ellis** both won recognition for their work this year in separate competitions.

Dr. Arasu, who has been pursuing a degree in veterinary medicine after completing a Ph.D. in parasitology and two postdoctoral fellowships in molecular biology, won first prize in the veterinary student competition of Cornell's Veterinary College Centennial Research Poster Day. Her poster described her work on recombinant *T. spiralis* proteins. Dr. Arasu has worked in this laboratory in her "spare time" and will receive her D.V.M. in June, 1994. She has

accepted a position as an assistant professor in the veterinary college of the Virginia Polytechnic Institute.

Lauri Ellis, a 1989 graduate of the Pennsylvania State University, is a former National Science Foundation Fellow and Andrew Dickson White Fellow. The latter is the most prestigious graduate fellowship awarded by Cornell University. This year, Ms. Ellis won a prize at the summer Gordon Conference on Molecular and Immunological Aspects of Parasitism for her poster on glycan targets of protective anti-*Trichinella* antibodies.

ALEXIS WENSKI-ROBERTS



Prema Arasu and Lauri Ellis

Equine Genetics Center

A

S A FETUS GROWS INSIDE THE UTERUS, it is shielded from most invading pathogens by its mother's immune system. But how does the uterus distinguish between undesirable foreign intruders and the developing fetus and placenta, whose paternally derived genes are also foreign?

It is not maternal recognition of the mother's half of its genes that ensures the survival of the fetus; even in surrogate mothers, whether humans, cattle, horses, or other mammalian species, there is no evidence that transferred embryos are attacked and destroyed by the mother's immune system. Once established, such

pregnancies appear to have the same success rate as normal pregnancies, even though the mother and fetus may have no genes in common. In extraordinary cases, pregnancy can succeed even when the surrogate mother and fetus are of two distinct species, such as zebra and horse.

Part of the solution to the riddle of the immunological survival of the developing fetus appears to

be in the particular types of white blood cells, or leukocytes, that inhabit the uterus. The uterus has developed a specialized cohort of immune cells that differ from those found circulating in blood or in the central organs of the immune system, such as the lymph nodes and

spleen. The uterine leukocytes even appear to be different from leukocytes of other components of the mucosal immune system, such as the gut or respiratory tract.

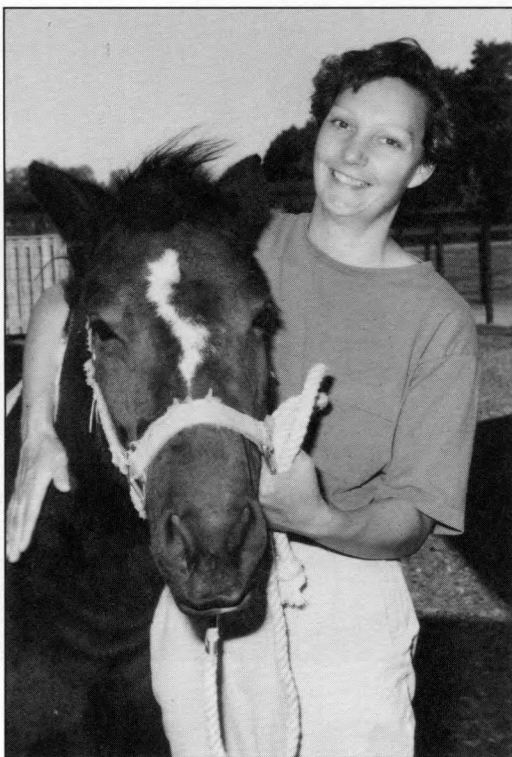
During the past six years a major effort of the Equine Genetics Center has been to characterize the distribution and function of leukocytes in the uterus of the mare, in both pregnant and non-pregnant states. An important component of this research was a workshop on horse leukocytes organized in 1991 by our laboratory and Dr. Julia Kydd of the Animal Health Trust in Newmarket, UK. The meeting brought together research scientists and veterinarians from around the world who had produced genetically engineered monoclonal antibodies capable of identifying and distinguishing sub-populations of horse leukocytes.

Gabriele Grünig, a German veterinarian who has been conducting research for her Ph.D. degree, has used the antibodies defined in the workshop to determine the composition of leukocytes in the uterus of the mare, and this has brought a surprising finding. Thus far her work appears to show a distribution of white blood cells in the uterus of the mare that is much more like that found in the blood and other central lymphoid or-

The uterus has developed a specialized cohort of immune cells that differ from those found circulating in blood or in the central organs of the immune system.



ALEXIS WENSKI-ROBERTS



Doug Antczak with Crowd Pleaser and her foal, Belle Tower (above), Jane Miller with Oreo (far left), and Sarah Deacon

gans than it is in humans and mice, two other well-studied species. A high proportion (50 to 80 percent) of uterine leukocytes of humans and rodents are of an

*Different species
may have found
distinct solutions to
the immunological
puzzle of pregnancy.*

unusual type known as large granular lymphocytes, while these cells appear to account for less than ten percent of the uterine leukocytes in the mare. This suggests that different species may have found distinct solutions to the immunological puzzle of pregnancy. The composition of uterine leukocytes may be correlated with, or even determined by, the type of placenta that has evolved in a particular species. In this regard the horse, cow, sheep, and pig all have primarily non-invasive placentas, while the placen-

tas of humans and rodents are invasive, burrowing deep into the uterus.

A second important finding was that the distribution of leukocytes that cluster around the endometrial cups seems to be different from the distribution of leukocytes in other parts of the pregnant and non-pregnant uterus. The endometrial cups, the small, invasive portion of the horse placenta, are intriguing for a number of reasons, one of which is that the cups are destroyed by the end of the first third of pregnancy, while the remainder of the placenta and fetus are left un-

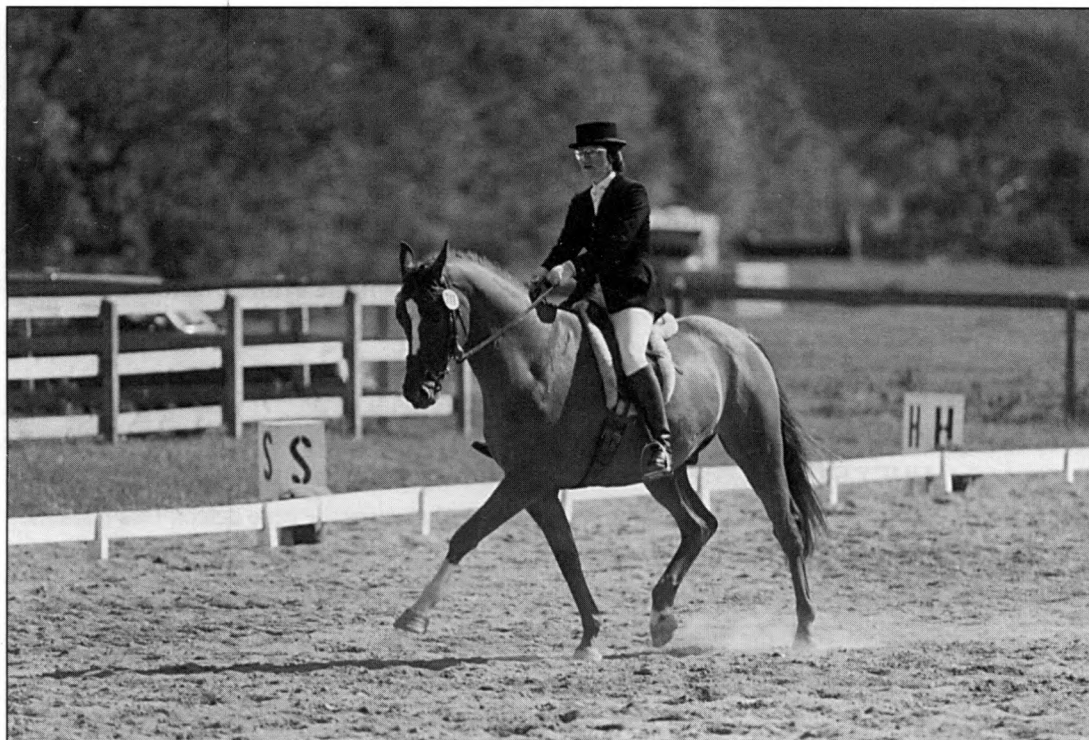
harmed. By studying the cytokines, or chemical messages, produced by equine uterine leukocytes we hope to shed light on the mechanism of destruction of the endometrial cups and on this fascinating type of mucosal immunity.

—D. F. Antczak

POSTER WINS PRIZE

Juli Maher, a veterinarian and Ph.D. student in the Equine Genetics Center, shared the first prize in the graduate student and veterinary resident section of the Centennial scientific poster competition sponsored by the College of Veterinary Medicine in October. Her poster described her studies of the molecular mechanisms by which the horse fetus prevents the expression of transplantation antigens on the surface of the placenta. This strategy greatly reduces the number of foreign paternal antigens exposed to the mother's immune system, and consequently also reduces the types of maternal immune responses that can be mounted. It is thought that these mechanisms are essential to the survival of the fetus.





Gabi Grünig (left) has established herself as one of the leading dressage competitors in upstate New York with her horse, Marais de Lully, a 12-year-old Swedish warmblood.



Jim Hardy with Bear (left) and Jessica Baker

Laboratory for the Study of Inherited Canine Reproductive Diseases



THE WORK OF OUR LABORATORY concerns inherited defects of the canine reproductive system. We continue to move from classical anatomic and microscopic studies to the use of molecular techniques in the characterization of persistent Müllerian duct syndrome and XX sex reversal, two genetic abnormalities in the development of the uterus and testes.

PERSISTENT MÜLLERIAN DUCT SYNDROME

Müllerian ducts, the tissue precursors of the uterus, develop in all canine embryos. As embryonic sexual differentiation proceeds, the male testis secretes a hormone, called Müllerian inhibiting substance (MIS), that binds to receptors on the cells of the Müllerian ducts and initiates their destruction. Persistent Müllerian duct syndrome, an inherited

defect of Miniature Schnauzers, involves a failure of this process that allows the Müllerian duct system in a genetic male to develop into a uterus. Our efforts to pinpoint the defect have previously shown that affected dogs produce MIS at the correct time to cause regression of the Müllerian duct system. Confocal microscopy studies are confirming our hypothesis that the MIS receptors in the ducts of

affected dogs are nonfunctional; therefore, it is likely that a mutation in the gene for the MIS receptors is the cause of the abnormality. Further studies will seek to isolate the receptor gene in nor-

mal and affected dogs in order to develop a practical method to identify animals that carry the gene defect. This research should also benefit humans, as MIS is being investigated as a potential therapeutic agent for some cancers by Patricia Donahoe and David MacLaughlin, our collaborators at Massachusetts General Hospital. Characterization of the mutation in the MIS receptor gene will also be helpful in determining which parts of the receptor are important to normal function.

XX SEX REVERSAL

This condition concerns the growth of testes in dogs that have two X chromosomes and are therefore genetic females. In addition to a uterus, affected dogs have incomplete testes that produce MIS very late in embryonic development. This abnormality occurs in seven different breeds, and we are studying it in American Cocker Spaniels and German Short-haired Pointers. We are currently looking for the genes that control testicular development and those that regulate production of MIS by the testis.

Virginia Fajt, a veterinary student from Auburn University who took part in Cornell's Summer Leadership Program, extracted DNA from affected dogs

It is likely that persistent Müllerian duct syndrome is caused by a mutation in the gene that regulates the development of the MIS receptors.

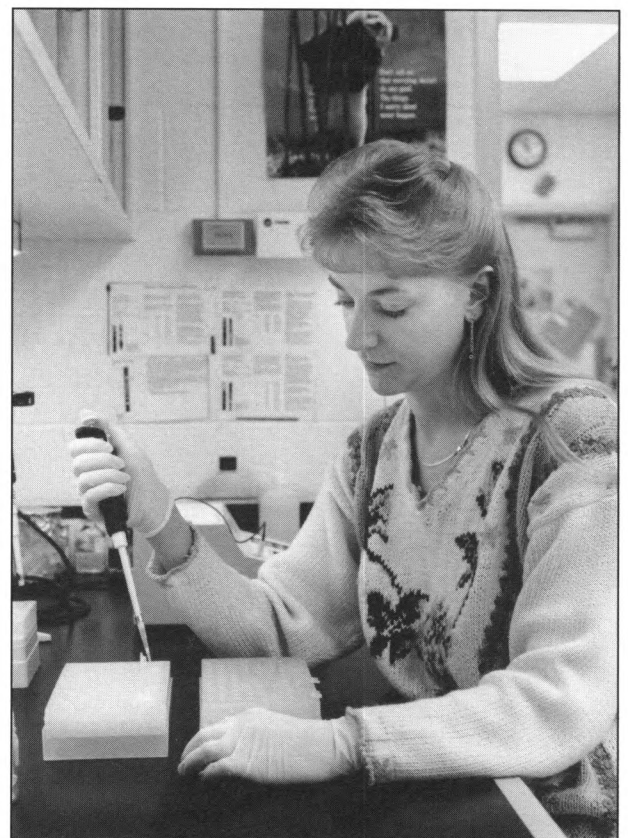


Vicki Meyers-Wallen with
Olga (left) and Vicky
Palmer (below)

for the study. Ina Dobrinski, a veterinarian and rotating graduate student, and Vicky Palmer examined the DNA of normal male dogs for the testis-determining gene. This gene has not yet been described in the dog but is located on the Y chromosome in other mammals. XX sex reversed dogs do not possess a Y chromosome. Our goal is to determine whether the testis-determining gene is located on a different chromosome in these animals and to develop a practical method of examining DNA to identify affected and carrier dogs. In this way we hope to gain a better understanding of the normal genetic controls on testis development in man and other mammals.

— Vicki Meyers-Wallen

*We are looking for
the genes that
control testicular
development and
those that regulate
production of the
hormone MIS.*



Donnelley Laboratory of Gene Regulation and Expression

M

UCOPOLYSACCHARIDOSIS (MPS) is a large group of inherited diseases with varying and often severe clinical manifestations.

Found in dogs, cats, mice, and humans, these diseases result from deficiencies of different lysosomal enzymes needed to break down mucopolysaccharides, or glycosaminoglycans, in tissues throughout the body.

The lack of any one of the lysosomal enzymes needed to remove specific carbohydrate portions of these large, complex molecules leads to the accumulation of undegraded glycosaminoglycans in the

lysosomal compartments of cells. Because glycosaminoglycans are present in all tissues, the effects of their abnormal accumulation are widespread and may include liver enlargement, bone or facial deformities, blindness, mental retardation, and early death.

Our laboratory is studying dogs with MPS VII, a syndrome caused by a deficiency of the lysosomal enzyme β -glucuronidase (GUSB). We have focused our

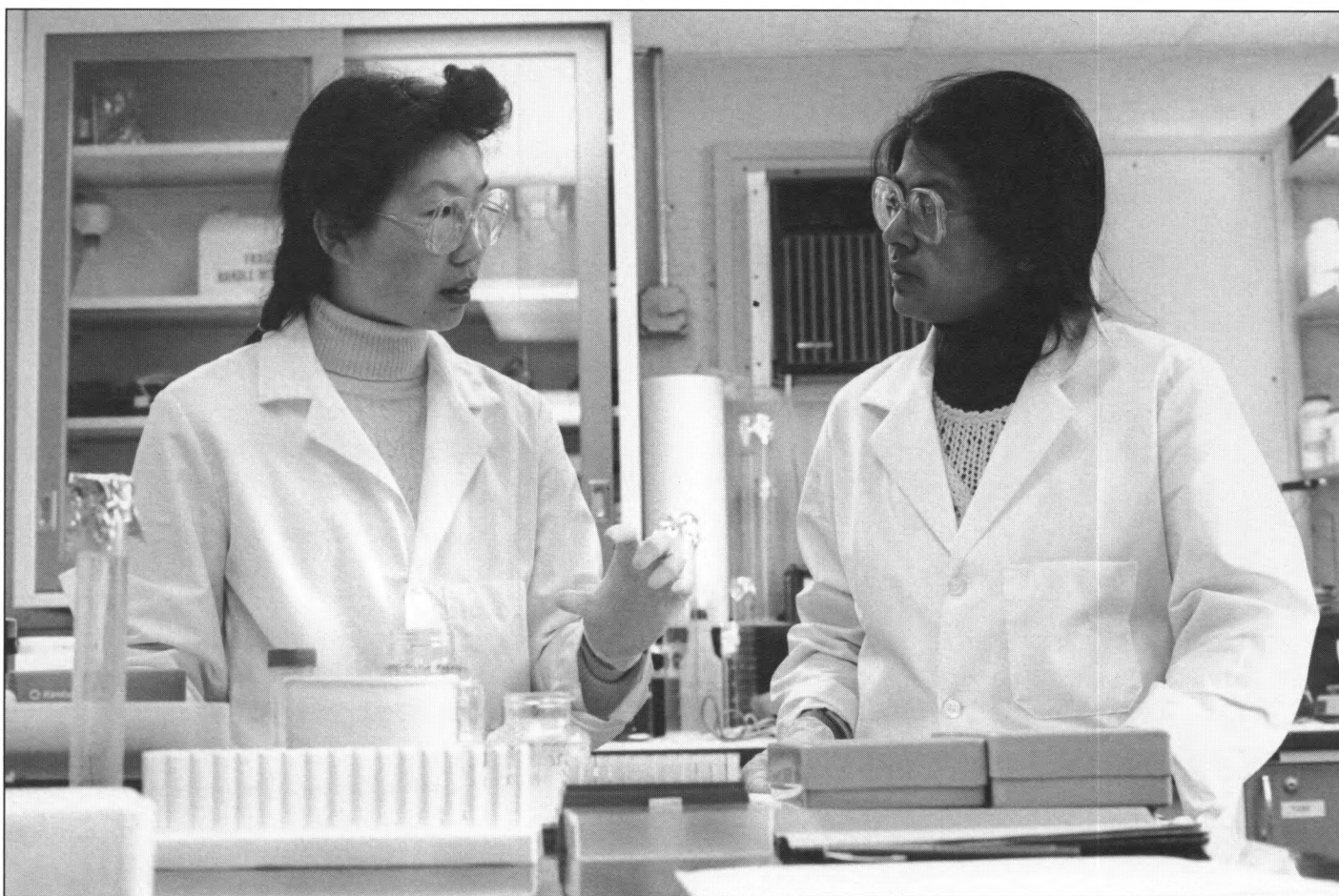
research on the effects of the GUSB deficiency on the retinal pigment epithelium of the eye, the site of the greatest lysosomal enzyme activity in the body. The build-up of glycosaminoglycans that re-

sults from insufficient GUSB production causes the cells of the retinal pigment epithelium to become hypertrophied, or thickened; this leads to the degeneration of the photoreceptors and ultimately to blindness.

Although the genetic defects that cause MPS VII have been characterized in some humans and mice, very little is known about the corresponding defects in dogs and cats. In studying samples provided by our colleague Mark Haskins of the University of Pennsylvania, we found that GUSB activity in the retinal pigment epithelium of diseased dogs was dramatically reduced—at most five percent of that in normal dogs. Our examination of the proteins present in the diseased retinal pigment epithelial cells showed a very low level of detectable GUSB. Having characterized the biochemical deficiency, we will next seek to identify the defect in the DNA that is responsible for the disease.

The striking and limited abnormalities present in the retinal pigment epithelium-photoreceptor interface make this an ideal experimental system to use in examining treatment strategies for this and other retinal diseases. We are collaborat-

Now that we have characterized the biochemical deficiency, we want to identify the defect in the DNA that is responsible for mucopolysaccharidosis VII in the dog.



ing with Gustavo Aguirre in an effort to treat MPS VII by transferring normal copies of the GUSB gene to diseased animals. We have begun the genetic manipulation of diseased canine retinal pigment epithelium in culture by infecting the cells with a harmless retrovirus engineered to carry normal copies of the GUSB gene cloned from rats. Our analysis of the infected cells showed that GUSB activity increased 80 to 100-fold as a result of treatment. The enzyme expressed in the treated cells was not only active but also targeted to the proper lysosome compartment. We are greatly encouraged by our success at the culture dish level

and will continue to work to develop appropriate means to treat patients with MPS VII.

This work profited from the contributions of Christopher Laing, a veterinary student from the University of Sydney, who helped characterize the disease while working as a summer fellow in our laboratory.

—Jharna Ray

*Yanggeng Wu and
Jharna Ray*

Inherited Eye Disease Studies Unit

THE TERM “progressive retinal atrophy” (PRA) describes a complex of inherited, blinding diseases of dogs that affect rods and cones, the light-gathering cells of the eye. During the past 25 years we have identified seven forms of PRA that, though clinically similar, are caused by separate gene defects. While one form of PRA, *prcd*, appears to be present in many breeds of dogs, several other disorders occur only in a single breed.

We have demonstrated conclusively that PRA in the Siberian Husky is an X-linked recessive disorder.

We have just achieved a breakthrough with one breed-specific disorder, rod-cone dysplasia 1 of Irish Setters. Following on work in our laboratory and elsewhere that identified the underlying biochemical and genetic defects, we have now developed a blood-based DNA test to differentiate normal, carrier, and affected status in Irish Setters. We will detail this work in a forthcoming

Institute newsletter and in the next annual report. In this report we summarize work done during the year on two other forms of PRA: XLPRA and *prcd*.

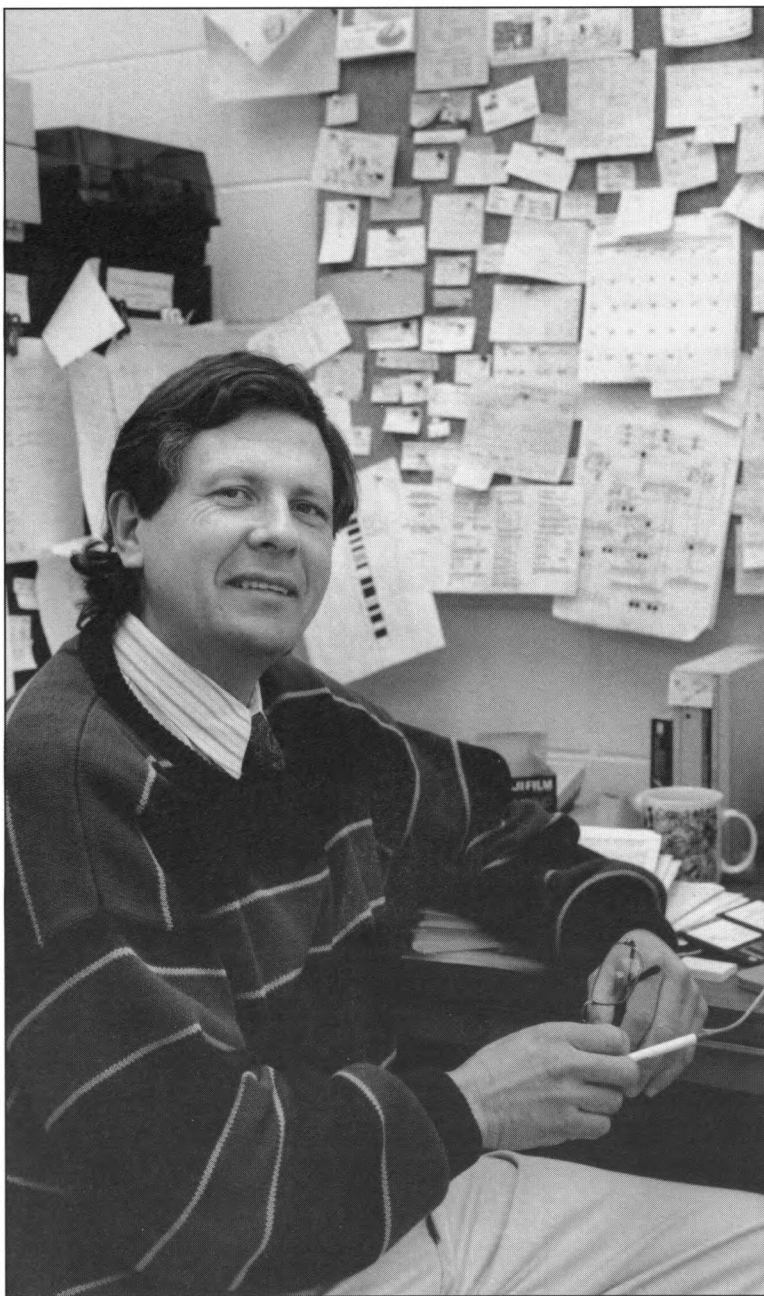
X-LINKED PROGRESSIVE RETINAL ATROPHY

X-linked PRA is inherited only in the Siberian Husky breed. The anecdotal observations of breeders and others had raised an intriguing possibility—that the defective gene causing the disease of Siberian Huskies might be carried on the X chromosome. If so, this would be the only form of PRA linked to the

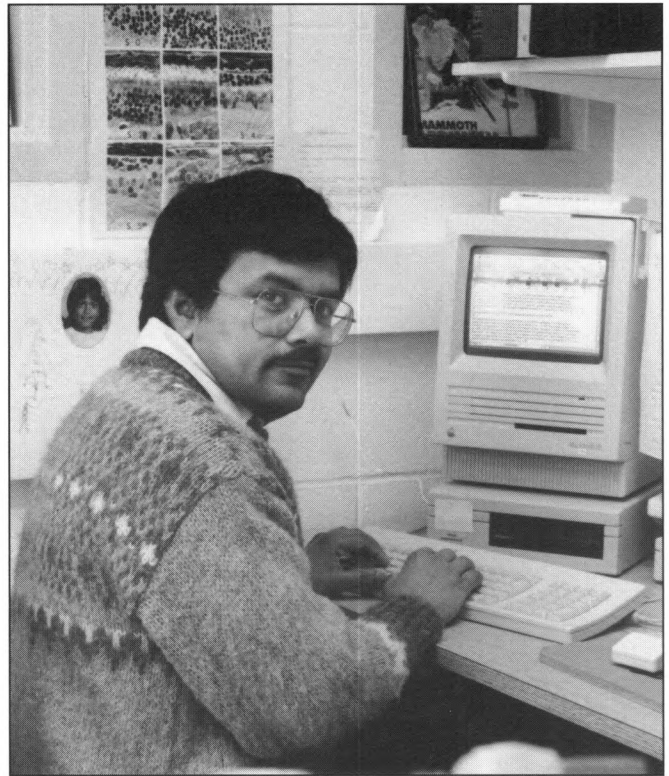
X chromosome. All other forms of PRA are autosomal recessive disorders.

The hallmark of X-linked recessive inheritance is the absence of male-to-male transmission of disease. Since male sex is determined by the inheritance of the father's Y chromosome, it is impossible for an X-linked disease to pass from father to son. Conversely, all daughters must inherit their father's X chromosome. If it contains a defect, all female offspring will necessarily carry that defect. The mother contributes one of her two X chromosomes to each of her offspring, regardless of sex. If one of the maternal X chromosomes carries a defective gene, offspring will have a 50–50 chance of inheriting the flawed copy. Sons that inherit an X-linked mutation from their mothers develop disease, while most daughters are clinically affected only when they acquire the gene defect from both parents. For this reason, X-linked recessive disorders are far more prevalent in males than in females.

In order to define the pattern of inheritance for the Siberian Husky disease, we began by breeding an affected male Siberian Husky to normal female Beagles. All offspring of the affected male Siberian Husky inherited a normal X chromo-

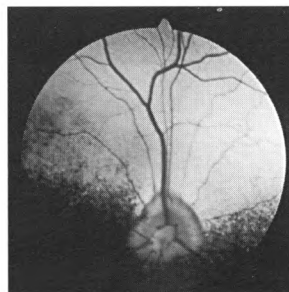


*Greg Acland (left) and
Kunal Ray (below)*

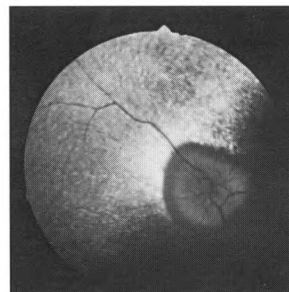


some from their genetically normal mothers. The results of those and subsequent breedings demonstrated conclusively that PRA in the Siberian Husky is an X-linked recessive disorder, which we have now designated as XLPRA.

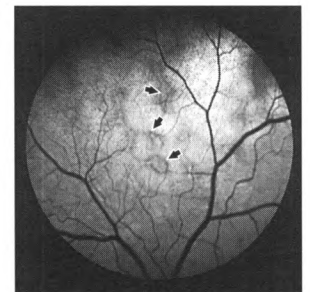
Affected dogs younger than two years of age can be recognized in a clinical setting by retinal thinning and loss of retinal blood vessels. Dogs examined between the ages of three and five years of age show more advanced disease, with disappearance of retinal blood vessels and retinal atrophy, as shown in the photographs at right. The end stage — blindness — occurs at about five or six years of age.



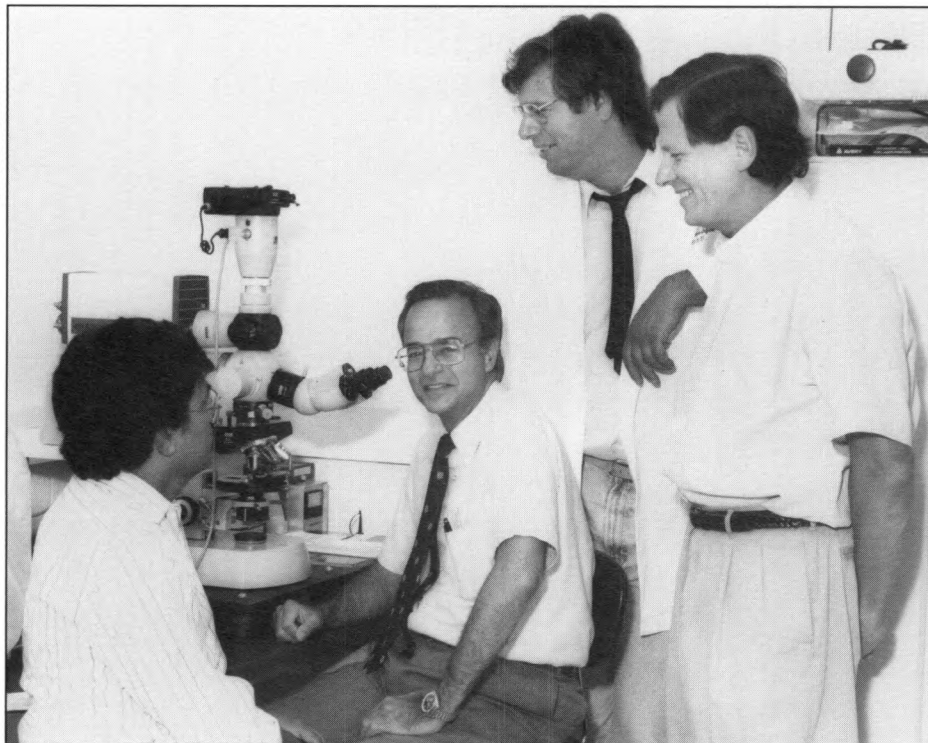
Retinal photographs of dogs.
A. Normal retina. Note that the retinal blood vessels are prominent.



B. PRA-affected retina, advanced disease. Retinal blood vessels are thin and few in number. This appearance of the retina is typical for the advanced stage of disease in all forms of PRA.



C. XLPRA carrier retina. The retinal blood vessels are normal, but there is a patchy retinal atrophy (arrows) that is characteristic of the disease in adult female carriers.



Shown left to right:
Kunal Ray, Gus Aguirre,
Ben Herschfield, and Greg
Acland.

Our long-term research objectives are to identify the gene responsible for XLPRA and to characterize the defect at the molecular level.

Achievement of these objectives will be essential to understanding how the mutant gene causes disease. XLPRA is thought to be a canine counterpart of a form of X-linked retinitis pigmentosa (XLRP) in humans; if so, the information we gain may be helpful in the development of strategies for therapy and prevention of the disorder in both dogs and man.

Three forms of XLRP have been identified in humans. All have been genetically mapped to the short arm of the X chromosome, but, to date, the genes have not been identified nor the defect characterized. With the help of human gene probes, we are attempting to map the gene that is defective in the XLPRA study pedigree. When the results of these studies are in hand, we will be able to establish the order of the genes on the canine X chromosome that correspond to the three human genes, establish whether

relative gene order has been evolutionarily conserved in the two species, and determine the relative map position of XLPRA.

PROGRESSIVE ROD-CONE DEGENERATION

Progressive rod-cone degeneration (*prcd*) is an especially widespread, autosomal recessive form of PRA. Like XLPRA, *prcd* appears to be the canine equivalent of a form of retinitis pigmentosa in humans. We have established through breeding studies that the identical gene is defective in Poodles, American and English Cocker Spaniels, and Labrador Retrievers, and

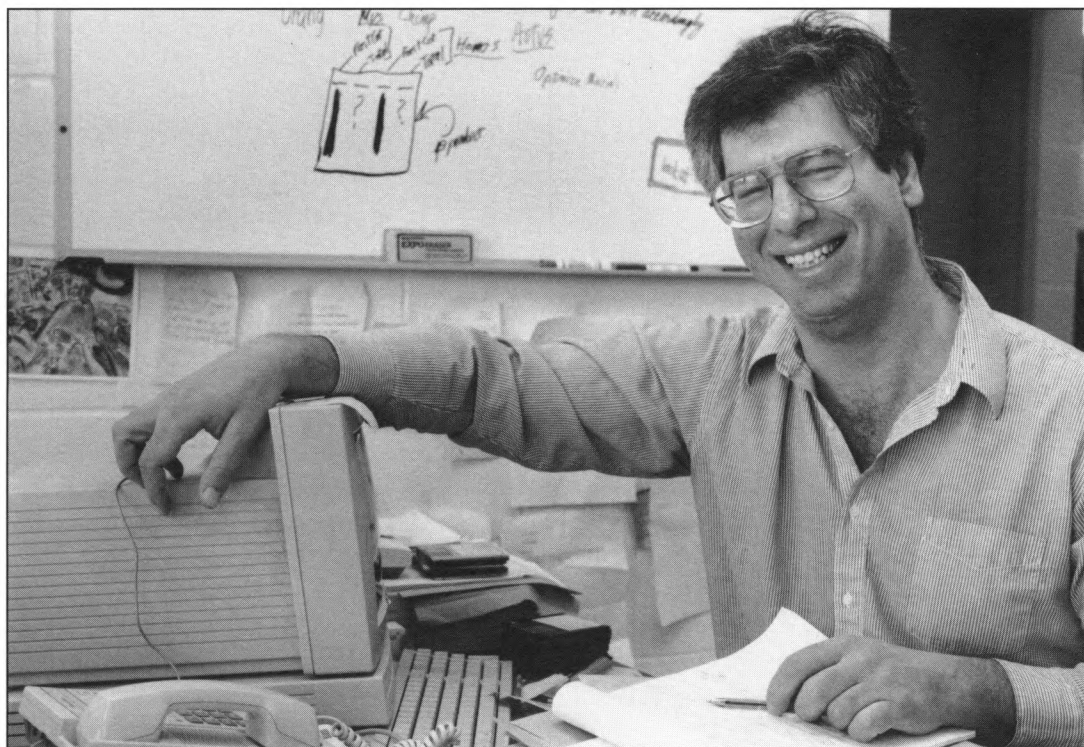
we suspect it to be the cause of late-onset PRA in many other breeds of dogs as well.

Our current studies center on the identification of a candidate gene for *prcd*. We have begun by examining opsin, the protein that catches light in the retina. The opsin gene has the highest level of expression in the mam-

malian photoreceptors, and mutations in the opsin gene have been causally associated with many cases of retinitis pigmentosa in humans. We have found that the level of opsin in the photoreceptors is normal in early disease but begins to decline once degeneration of the rods and cones begins. Although opsin is initially abundant in the retinas of *prcd*-affected dogs, we are interested in learning whether the onset of disease results from defects in the structure of the protein.

Our parallel approach has been to examine the coding region of the opsin

*We expect to know
within a year whether
a defect in the
opsin gene causes
progressive rod-cone
degeneration.*



Ben Hershfield (left),
Katy Gropp (middle),
Vicky Baldwin
(bottom left), and Sue
Pearce-Kelling

gene using powerful and highly sensitive new methods of scanning genes for mutations. The technique, called single or double-stranded conformational polymorphism, allows us to detect any changes that alter the structure of a gene. We wish to determine if mutations are present that could affect the gene sequence and protein structure without affecting the quantity of the protein that is produced. Our examination of the opsin gene has been simplified by the recent elucidation of the gene's DNA sequence in the dog, and we expect to know within a year whether the opsin gene is defective in *prcd*.

The year 1993 was a memorable one for our laboratory. For those of us who made the move from the University of Pennsylvania, it was a year to get settled in Ithaca and in our superb new laboratory facilities. For the Institute's "old" staff, it was a lot of hard work, and we would like to thank everyone whose extra efforts eased our transition and made us feel so welcome.

—Gustavo Aguirre, Gregory Acland,
Bennett Hershfield, and Kunal Ray



Publications

The following is a list of manuscripts published by staff members of the Baker Institute in 1993. Publications listed as "in press" in last year's report are repeated this year, with their original numbers, to record their full bibliographical details.

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Special Memorials

FRANK BLOOM

Frank Bloom, D.V.M. '30, a practicing veterinarian credited with creating the field of small animal clinical pathology, died July 9 in Hallandale, Florida.

During forty years in successful private practice in Flushing, New York, Dr. Bloom pursued the study of canine and feline pathology both as a means to improved diagnosis and out of a lively sense of scientific curiosity. His interests were wide-ranging and yielded articles and textbooks of fundamental importance to veterinary pathology. His work to develop a practical method for obtaining bone marrow from dogs for clinical use was recognized with the 1964 AVMA Practitioner Research Award. When Dr. Bloom was inducted in 1984 as a Distinguished Member of the American College of Veterinary Pathologists, of which he was a charter member, he was lauded as "the pioneer veterinary clinical pathologist in the United States." In 1988 he was awarded the Peter Olafson Medal for Lifetime Achievement in Veterinary Pathology.

Dr. Bloom was an Institute founder and a faithful contributor to the Cornell Research Laboratory for Diseases of Dogs. We are grateful to him for the very generous provision he made in his will to further canine health research at the Baker Institute.

CHARLES E. "TED" FLETCHER

Charles E. "Ted" Fletcher, '31 D.V.M. '33, an Institute founder, James Law Fellow, and the first recipient of the Institute's Founders' Award, died on New Year's Day. Dr. Fletcher, who was the New York State Veterinary Medical Society's Veterinarian of the Year for 1983, owned the Abbey

Veterinary Hospital in Riverdale, New York. Among his many activities, he wrote a daily pet column for the *New York Mirror* for six years during the 1950s. Dr. Fletcher headed the New York City Veterinary Medical Society in 1952 and served as president of the state society in 1961. He was an exceptionally generous sponsor of Institute research for the benefit of dogs. The following friends and associates have made gifts to the Institute in his memory:

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GEORGE A. GOODE

George A. Goode, D.V.M. '45, a founder and longtime supporter of the Institute's Cornell Research Laboratory for Diseases of Dogs, died August 21 at the age of 79. Dr. Goode built the Riverhead Animal Hospital in 1946 and practiced there until his retirement in 1975. He was active in local affairs and served as president of the Rotary Club and the Long Island Veterinary Medical Association. The following people have honored his memory with a gift to the Institute:

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 Yellow Springs Veterinary Clinic

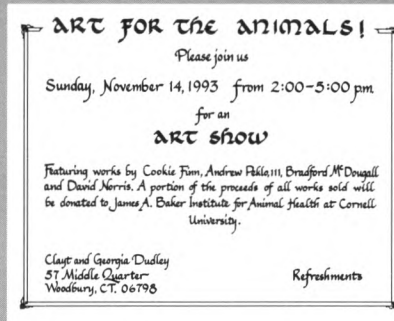
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Georgia and G. Clayton Dudley,
 D.V.M. '64, hosted an art show and sale at their veterinary hospital to benefit the Baker Institute. Thanks to the generosity of featured artists Cookie Finn, Andrew Peklo, III, Bradford McDougall, and David Norris as well as several patrons who kicked in something extra, Dr. and Mrs. Dudley's inspired initiative raised almost \$1,000 for our animal health research programs.

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 Dina Broudny (in memory of Amber and Bea Bea)
 Charles E. Brown, D.V.M. (in memory of Reflex)
 Mr. and Mrs. Kolman Brown (in memory of Desi)
 Mr. and Mrs. Richard C. Brown (in memory of Molly)
 Dr. and Mrs. Ronald Brown (in memory of Maggie, Milo, and Jasmine)
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 Mary Ann Ciccarelli (in memory of Malcolm)
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“ Once again, it is my wife's and my pleasure to send the James A. Baker Institute a small token of our appreciation for the research work you are pursuing. This check is in memory of our three beloved, four-footed buddies, Rex, Dusty, and Ginger. If you will arrange to have an associate process the accompanying matching gift form from my employer, the check will be double-matched.

I have mentioned in a previous letter that it was your parvovirus vaccination that saved the life of our Great Dane, Ginger. Perhaps our remembrance will encourage someone to take the extra step in research that could result in another breakthrough discovery.

Steven & Arlene Grey, N. Massapequa, NY

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“ We were very touched and proud that the Keating Animal Hospital made a contribution to your work in the name of our wonderful dog, Bonhomme. It is a very heartening gesture for us to know that Dr. Keating thinks enough of pets and their “families” to honor the relationship. All dogs, like all of us humans, should have care and love and dignity. We are happy to send a contribution to help you continue the work on helping future pets and future “families.”

We shall remember Bonhomme always. We shall honor that companionship by getting another dog—or maybe two. . . it is sure that pets make us humans more decent and, finally, more human.

Martha W. Coigney, New York, NY

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 Louise F. Lamont
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 Mr. and Mrs. Myron B. Lampert (in memory of Princess)

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 Mr. and Mrs. Gene Lane (in memory of Heidi)
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 Mr. and Mrs. Peter Lange
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“ Our internal medicine veterinarian, Dr. William Rogers of Milford, Ohio, speaks highly of your organization and its various research programs. We are especially pleased by the fact that our money goes to fund projects that benefit pet health rather than to research programs whose main thrust is their impact on human health, with benefits to pets a secondary importance. I am sure research programs which focus from the beginning on pets are going to have a greater impact on the future quality of life for cats and dogs.

The importance of your work is appreciated by all of us who love animals. We continue to wish you every success.

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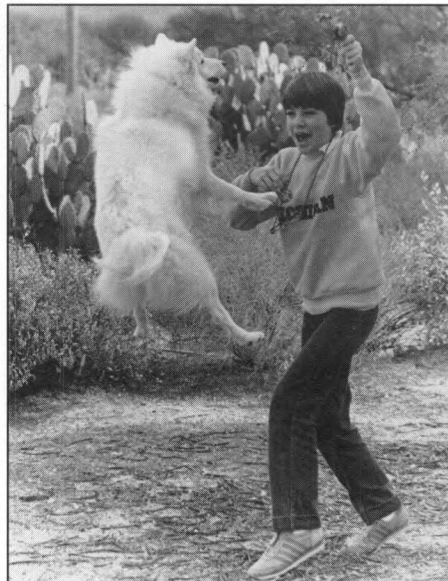
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Sage was only 4 1/2 years old, and those who loved her will bear a Bouvier-shaped hole in their lives. You probably hear testimonies all the time about "my wonderful Collie" and "our beloved Poodle" so I will not add ours to the pile. They are meaningful only to those who know and love the dogs. But know this: every word is true. We all feel that way about our dogs, and everyone who ever met Sage felt that way, too, including the four vets at Manchester Vet, at least two of whom I know were educated at Cornell. So know two more things: one, that at least two of your grads have our utmost admiration and gratitude for the professional and compassionate treatment shown our Sage; and two, that those of us who love these dogs and would have done anything to see them well, deeply appreciate and support the work done by you to prevent and cure the diseases that take them too young.



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“ I was surprised and very touched to receive your letter yesterday telling me about the donation made by Dr. Rogers, Dr. Harris, and Dr. Apt in memory of my Tory. I sincerely appreciate their thoughtfulness.

Your letter also made me aware that I should make a similar contribution. I was going to place a memorial ad on him in our Specialty catalog, but in truth, the money is better spent to benefit the lives of other dogs...

Please accept this small contribution in memory of Tory, Ch. Sundaze Victory in Motion. I hope that it will help so that some time in the future, other dogs can be free of this and other devastating diseases.

Pamela G. Rightmyer, Las Vegas, NV

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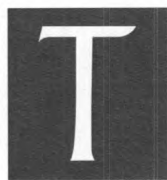
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Ways of Giving



THE CORNELL UNIVERSITY Board of Trustees, in establishing the Baker Institute, authorized the Treasurer's Office of Cornell to be custodian of all funds given in support of the Institute.

You are thus assured that your gift will have the maximum benefit. There are many ways you can give to advance the work of the Baker Institute. Some of these opportunities offer substantial income tax and estate tax benefits.

Checks All checks should be made payable to the Baker Institute and mailed to:

Office of Public Affairs
James A. Baker Institute for
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Cornell University
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Appreciated Stocks Selling appreciated stocks is almost certain to increase your taxes, but if you give the stocks to Cornell outright and deduct their full current market value as a charitable contribution, you can probably avoid all or most of the capital gains tax. To complete the transaction with maximum speed and at lowest cost:

1. take the certificate to your bank or broker;
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3. instruct your bank or broker to telephone the *Office of Trusts and Estates* at 607 256-5600; and
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Depreciated Stocks You get a maximum benefit from a gift of depreciated stocks by selling them and giving the cash to Cornell. That way you get the capital loss allowance and a charitable contribution deduction for the amount of the gift. Instruct your bank or broker to sell the shares or securities and send the proceeds as a gift to Cornell for the James A. Baker Institute for Animal Health.

Bequests Charitable bequests provide substantial estate tax benefits. They can be gifts of land or buildings, securities, personal property, or cash. The Cornell University counsel suggests the following provision: *I hereby give, devise, and bequeath [description of property] to Cornell University, an educational corporation located at Ithaca, New York, for the uses and purposes of the James A. Baker Institute for Animal Health.*

Deferred Giving An income-producing trust enables you to make a gift to the Baker Institute, gain income for life, and derive tax benefits. A beneficiary may be named to receive the income, too. The Institute offers three plans: the *Pooled Life Income Fund*, the *Annuity Trust*, and the *Unitrust*. You may also establish a *Charitable Lead Trust*, which will first pay income to Cornell and later transfer assets back to you or a third transfer assets back to you or a third party. Financial planning involving deferred gifts requires expert advice from your attorney and other specialists. If you are interested in deferred giving, please notify the Office of Public Affairs and we will make arrangements for you to receive more information.

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